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* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 14:19:55 ON 02 MAY 2006

=> fil .bec
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILES 'MEDLINE, SCISEARCH, LIFESCI, BIOTECHDS, BIOSIS, EMBASE, HCAPLUS, NTIS,
       ESBIOBASE, BIOTECHNO, WPIDS' ENTERED AT 14:20:25 ON 02 MAY 2006
ALL COPYRIGHTS AND RESTRICTIONS APPLY.  SEE HELP USAGETERMS FOR DETAILS.

11 FILES IN THE FILE LIST

=> s sulfotransferase#
FILE 'MEDLINE'
L1         2513 SULFOTRANSFERASE#

FILE 'SCISEARCH'
L2         3100 SULFOTRANSFERASE#

FILE 'LIFESCI'
L3          743 SULFOTRANSFERASE#

FILE 'BIOTECHDS'
L4          160 SULFOTRANSFERASE#

FILE 'BIOSIS'
L5         3382 SULFOTRANSFERASE#

FILE 'EMBASE'
L6         2715 SULFOTRANSFERASE#

FILE 'HCAPLUS'
L7         3819 SULFOTRANSFERASE#

FILE 'NTIS'
L8           18 SULFOTRANSFERASE#

FILE 'ESBIOBASE'
L9         1176 SULFOTRANSFERASE#

FILE 'BIOTECHNO'
L10        1049 SULFOTRANSFERASE#

FILE 'WPIDS'
L11         141 SULFOTRANSFERASE#

TOTAL FOR ALL FILES
L12       18816 SULFOTRANSFERASE#

=> s l12 and (phenol? or pst)
FILE 'MEDLINE'
          39900 PHENOL?
           1916 PST
L13         510 L1 AND (PHENOL? OR PST)

FILE 'SCISEARCH'
          77266 PHENOL?
           2474 PST
L14         780 L2 AND (PHENOL? OR PST)

FILE 'LIFESCI'
          19000 PHENOL?
```

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           750 PST
L15        174 L3 AND (PHENOL? OR PST)

FILE 'BIOTECHDS'
         12465 PHENOL?
           181 PST
L16         17 L4 AND (PHENOL? OR PST)

FILE 'BIOSIS'
         73196 PHENOL?
          2858 PST
L17        649 L5 AND (PHENOL? OR PST)

FILE 'EMBASE'
         35451 PHENOL?
          1599 PST
L18        606 L6 AND (PHENOL? OR PST)

FILE 'HCAPLUS'
        411213 PHENOL?
          2887 PST
L19        792 L7 AND (PHENOL? OR PST)

FILE 'NTIS'
          6655 PHENOL?
           132 PST
L20          0 L8 AND (PHENOL? OR PST)

FILE 'ESBIOBASE'
         31316 PHENOL?
           632 PST
L21        228 L9 AND (PHENOL? OR PST)

FILE 'BIOTECHNO'
         10046 PHENOL?
           687 PST
L22        221 L10 AND (PHENOL? OR PST)

FILE 'WPIDS'
        125674 PHENOL?
           502 PST
L23         10 L11 AND (PHENOL? OR PST)

TOTAL FOR ALL FILES
L24       3987 L12 AND (PHENOL? OR PST)

=> s l24 and (muta? or allel? or variant# or allozym? or polymorph?)
FILE 'MEDLINE'
        514173 MUTA?
        111876 ALLEL?
        112552 VARIANT#
          1716 ALLOZYM?
        157724 POLYMORPH?
L25        103 L13 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'SCISEARCH'
        499963 MUTA?
        105566 ALLEL?
        125062 VARIANT#
          7697 ALLOZYM?
        186405 POLYMORPH?
L26        162 L14 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'LIFESCI'
        226894 MUTA?
```

```
         50960 ALLEL?
         38010 VARIANT#
          4391 ALLOZYM?
         63310 POLYMORPH?
L27         28 L15 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'BIOTECHDS'
         45819 MUTA?
          8130 ALLEL?
         15666 VARIANT#
            36 ALLOZYM?
          9304 POLYMORPH?
L28         10 L16 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'BIOSIS'
        557139 MUTA?
        127111 ALLEL?
        114609 VARIANT#
          8971 ALLOZYM?
        193949 POLYMORPH?
L29        102 L17 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'EMBASE'
        430411 MUTA?
         90195 ALLEL?
         98334 VARIANT#
          1080 ALLOZYM?
        135034 POLYMORPH?
L30         90 L18 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'HCAPLUS'
        524481 MUTA?
        103410 ALLEL?
        111626 VARIANT#
          2948 ALLOZYM?
        185843 POLYMORPH?
L31        144 L19 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'NTIS'
         10096 MUTA?
           612 ALLEL?
          4656 VARIANT#
            29 ALLOZYM?
          1634 POLYMORPH?
L32          0 L20 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'ESBIOBASE'
        263600 MUTA?
         59081 ALLEL?
         47253 VARIANT#
          3672 ALLOZYM?
         70320 POLYMORPH?
L33         69 L21 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'BIOTECHNO'
        242571 MUTA?
         55235 ALLEL?
         41198 VARIANT#
          1661 ALLOZYM?
         71286 POLYMORPH?
L34         63 L22 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

FILE 'WPIDS'
         29218 MUTA?
          7408 ALLEL?
```

```
         27723 VARIANT#
             7 ALLOZYM?
          8470 POLYMORPH?
L35          3 L23 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

TOTAL FOR ALL FILES
L36        774 L24 AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR POLYMORPH?)

=> s l36 not 1999-2006/py
FILE 'MEDLINE'
       4012402 1999-2006/PY
                 (19990000-20069999/PY)
L37         50 L25 NOT 1999-2006/PY

FILE 'SCISEARCH'
       7680103 1999-2006/PY
                 (19990000-20069999/PY)
L38         59 L26 NOT 1999-2006/PY

FILE 'LIFESCI'
        783533 1999-2006/PY
L39         16 L27 NOT 1999-2006/PY

FILE 'BIOTECHDS'
        158619 1999-2006/PY
L40          1 L28 NOT 1999-2006/PY

FILE 'BIOSIS'
       3944710 1999-2006/PY
L41         52 L29 NOT 1999-2006/PY

FILE 'EMBASE'
       3531731 1999-2006/PY
L42         47 L30 NOT 1999-2006/PY

FILE 'HCAPLUS'
       7447348 1999-2006/PY
L43         54 L31 NOT 1999-2006/PY

FILE 'NTIS'
        132438 1999-2006/PY
L44          0 L32 NOT 1999-2006/PY

FILE 'ESBIOBASE'
       2165128 1999-2006/PY
L45         34 L33 NOT 1999-2006/PY

FILE 'BIOTECHNO'
        611346 1999-2006/PY
L46         39 L34 NOT 1999-2006/PY

FILE 'WPIDS'
       6197246 1999-2006/PY
L47          0 L35 NOT 1999-2006/PY

TOTAL FOR ALL FILES
L48        352 L36 NOT 1999-2006/PY

=> dup rem l48
PROCESSING COMPLETED FOR L48
L49         103 DUP REM L48 (249 DUPLICATES REMOVED)

=> d tot

L49  ANSWER 1 OF 103     MEDLINE on STN                  DUPLICATE 1
```

TI The sulfuryl transfer mechanism. Crystal structure of a vanadate complex of estrogen **sulfotransferase** and **mutational** analysis.
SO The Journal of biological chemistry, (1998 Oct 16) Vol. 273, No. 42, pp. 27325-30.
Journal code: 2985121R. ISSN: 0021-9258.
AU Kakuta Y; Petrotchenko E V; Pedersen L C; Negishi M
AN 1998438504 MEDLINE

L49 ANSWER 2 OF 103 HCAPLUS COPYRIGHT 2006 ACS on STN
TI Rat, but not human, **sulfotransferase** activates a tamoxifen metabolite to produce DNA adducts and gene **mutations** in bacteria and mammalian cells in culture
SO Carcinogenesis (1998), 19(10), 1709-1713
CODEN: CRNGDP; ISSN: 0143-3334
AU Glatt, Hansruedi; Davis, Warren; Meinl, Walter; Hermersdorfer, Heino; Venitt, Stan; Phillips, David H.
AN 1998:701419 HCAPLUS
DN 130:50358

L49 ANSWER 3 OF 103 MEDLINE on STN DUPLICATE 2
TI A single amino acid, glu146, governs the substrate specificity of a human dopamine **sulfotransferase**, SULT1A3.
SO Molecular pharmacology, (1998 Dec) Vol. 54, No. 6, pp. 942-8.
Journal code: 0035623. ISSN: 0026-895X.
AU Dajani R; Hood A M; Coughtrie M W
AN 1999074339 MEDLINE

L49 ANSWER 4 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Human histamine N-methyltransferase pharmacogenetics: Common genetic **polymorphisms** that alter activity
SO MOLECULAR PHARMACOLOGY, (APR 1998) Vol. 53, No. 4, pp. 708-717.
ISSN: 0026-895X.
AU Preuss C V; Wood T C; Szumlanski C L; Raftogianis R B; Otterness D M; Girard B; Scott M C; Weinshilboum R M (Reprint)
AN 1998:312503 SCISEARCH

L49 ANSWER 5 OF 103 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
AN 1998212868 ESBIOBASE
TI Molecular cloning, expression, and functional characterization of novel mouse **sulfotransferases**
AU Sakakibara Y.; Yanagisawa K.; Takami Y.; Nakayama T.; Suiko M.; Liu M.-C.
CS Y. Sakakibara, Department of Biochemistry, University of Texas Health Center, Tyler, TX 75710, United States.
SO Biochemical and Biophysical Research Communications, (29 JUN 1998), 247/3 (681-686), 26 reference(s)
CODEN: BBRCA0 ISSN: 0006-291X
DT Journal; Article
CY United States
LA English
SL English

L49 ANSWER 6 OF 103 MEDLINE on STN DUPLICATE 3
TI A BseRI PCR/RFLP in an intron of the canine **phenol sulfotransferase** gene.
SO Animal genetics, (1998 Aug) Vol. 29, No. 4, pp. 329.
Journal code: 8605704. ISSN: 0268-9146.
AU Liu P C; Shibuya H; Nonneman D; Katz M L; Johnson G S
AN 1998418204 MEDLINE

L49 ANSWER 7 OF 103 MEDLINE on STN DUPLICATE 4
TI Expression of human estrogen **sulfotransferase** in Salmonella typhimurium: differences between hHST and hEST in the enantioselective activation of 1-hydroxyethylpyrene to a **mutagen.**

SO Chemico-biological interactions, (1998 Feb 20) Vol. 109, No. 1-3, pp. 249-53.
Journal code: 0227276. ISSN: 0009-2797.
AU Hagen M; Pabel U; Landsiedel R; Bartsch I; Falany C N; Glatt H
AN 1998226401 MEDLINE

L49 ANSWER 8 OF 103 MEDLINE on STN DUPLICATE 5
TI Genetic **polymorphisms** in human liver **phenol sulfotransferases** involved in the bioactivation of N-hydroxy derivatives of carcinogenic arylamines and heterocyclic amines.
SO Chemico-biological interactions, (1998 Feb 20) Vol. 109, No. 1-3, pp. 237-48.
Journal code: 0227276. ISSN: 0009-2797.
AU Ozawa S; Tang Y M; Yamazoe Y; Kato R; Lang N P; Kadlubar F F
AN 1998226400 MEDLINE

L49 ANSWER 9 OF 103 MEDLINE on STN DUPLICATE 6
TI Molecular biology of the human **phenol sulfotransferase** gene family.
SO The Journal of experimental zoology, (1998 Sep-Oct 1) Vol. 282, No. 1-2, pp. 223-30.
Journal code: 0375365. ISSN: 0022-104X.
AU Dooley T P
AN 1998390620 MEDLINE

L49 ANSWER 10 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI **Sulfotransferase**-mediated activation of **mutagens** studied using heterologous expression systems
SO CHEMICO-BIOLOGICAL INTERACTIONS, (20 FEB 1998) Vol. 109, No. 1-3, pp. 195-219.
ISSN: 0009-2797.
AU Glatt H (Reprint); Bartsch I; Christoph S; Coughtrie M W H; Falany C N; Hagen M; Landsiedel R; Pabel U; Phillips D H; Seidel A; Yamazoe Y
AN 1998:249180 SCISEARCH

L49 ANSWER 11 OF 103 MEDLINE on STN DUPLICATE 7
TI A review of the effects of manipulation of the cysteine residues of rat aryl **sulfotransferase** IV.
SO Chemico-biological interactions, (1998 Feb 20) Vol. 109, No. 1-3, pp. 107-16. Ref: 21
Journal code: 0227276. ISSN: 0009-2797.
AU Marshall A D; Darbyshire J F; McPhie P; Jakoby W B
AN 1998226389 MEDLINE

L49 ANSWER 12 OF 103 MEDLINE on STN DUPLICATE 8
TI **Sulfotransferase**-mediated genotoxicity of propane 2-nitronate in cultured ovine seminal vesicle cells.
SO Mutation research, (1998 Feb 23) Vol. 413, No. 1, pp. 69-81.
Journal code: 0400763. ISSN: 0027-5107.
AU Kreis P; Degen G H; Andrae U
AN 1998265570 MEDLINE

L49 ANSWER 13 OF 103 MEDLINE on STN DUPLICATE 9
TI On the nature of rat hepatic and mouse olfactory **sulfotransferases**.
SO Chemico-biological interactions, (1998 Feb 20) Vol. 109, No. 1-3, pp. 69-80. Ref: 22
Journal code: 0227276. ISSN: 0009-2797.
AU Matsui M; Tamura H; Nagai F; Homma H; Miyawaki A; Mikoshiba K
AN 1998226386 MEDLINE

L49 ANSWER 14 OF 103 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
AN 1998191947 ESBIOBASE

TI Molecular cloning, expression, and characterization of a novel mouse liver SULT1B1 **sulfotransferase**
AU Saeki Y.; Sakakibara Y.; Araki Y.; Yanagisawa K.; Suiko M.; Nakajima H.; Liu M.-C.
CS M.-C. Liu, Department of Biochemistry, University of Texas Health Center, PO Box 2003, Tyler, TX 75710, United States.
E-mail: liu@uthct.edu
SO Journal of Biochemistry, (1998), 124/1 (55-64), 43 reference(s)
CODEN: JOBIAO ISSN: 0021-924X
DT Journal; Article
CY Japan
LA English
SL English

L49 ANSWER 15 OF 103 MEDLINE on STN DUPLICATE 10
TI **Polymorphisms** of N-acetyltransferases, glutathione S-transferases, microsomal epoxide hydrolase and **sulfotransferases** : influence on cancer susceptibility.
SO Recent results in cancer research. Fortschritte der Krebsforschung. Progres dans les recherches sur le cancer, (1998) Vol. 154, pp. 47-85. Ref: 178
Journal code: 0044671. ISSN: 0080-0015.
AU Hengstler J G; Arand M; Herrero M E; Oesch F
AN 1999151055 MEDLINE

L49 ANSWER 16 OF 103 MEDLINE on STN DUPLICATE 11
TI Novel **sulfotransferases** cloned by RT-PCR: real proteins or PCR artifacts?.
SO Chemico-biological interactions, (1998 Feb 20) Vol. 109, No. 1-3, pp. 43-52.
Journal code: 0227276. ISSN: 0009-2797.
AU Gaedigk A; Lekas P; Berchuk M; Grant D M
AN 1998226384 MEDLINE

L49 ANSWER 17 OF 103 MEDLINE on STN DUPLICATE 12
TI Cloning of the human **phenol sulfotransferase** gene family: three genes implicated in the metabolism of catecholamines, thyroid hormones and drugs.
SO Chemico-biological interactions, (1998 Feb 20) Vol. 109, No. 1-3, pp. 29-41. Ref: 37
Journal code: 0227276. ISSN: 0009-2797.
AU Dooley T P
AN 1998226383 MEDLINE

L49 ANSWER 18 OF 103 LIFESCI COPYRIGHT 2006 CSA on STN
TI A BseRI PCR/RFLP in an intron of the canine **phenol sulfotransferase** gene
SO Anim. Genet., (19980800) vol. 29, no. 4.
ISSN: 0268-9146.
AU Liu, P.-C.; Shibuya, H.; Nonneman, D.; Katz, M.L.; Johnson, G.S.
AN 1999:2688 LIFESCI

L49 ANSWER 19 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI **Mutational** analysis of domain II of flavonol 3-**sulfotransferase**
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1 AUG 1997) Vol. 247, No. 3, pp. 1056-1062.
ISSN: 0014-2956.
AU Marsolais F (Reprint); Varin L
AN 1997:617594 SCISEARCH

L49 ANSWER 20 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Platelet sulphotransferase activity, plasma sulphate levels and sulphation

capacity in patients with migraine and tension headache
SO CEPHALALGIA, (NOV 1997) Vol. 17, No. 7, pp. 761-764.
ISSN: 0333-1024.
AU Alam Z (Reprint); Coombes N; Waring R H; Williams A C; Steventon G B
AN 1997:854716 SCISEARCH

L49 ANSWER 21 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Genetic and environmental factors associated with variation of human xenobiotic glucuronidation and sulfation
SO ENVIRONMENTAL HEALTH PERSPECTIVES, (JUN 1997) Vol. 105, Supp. [4], pp. 739-747.
ISSN: 0091-6765.
AU Burchell B (Reprint); Coughtrie M W H
AN 1997:575191 SCISEARCH

L49 ANSWER 22 OF 103 MEDLINE on STN DUPLICATE 13
TI Multiplicity of **sulfotransferases.**
SO Yakugaku zasshi : Journal of the Pharmaceutical Society of Japan, (1997 Nov) Vol. 117, No. 10-11, pp. 729-38. Ref: 34
Journal code: 0413613. ISSN: 0031-6903.
AU Matsui M
AN 1998076240 MEDLINE

L49 ANSWER 23 OF 103 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Genetic **polymorphisms** in human liver and colon **sulfotransferases** (SULTs) involved in the bioactivation of carcinogenic N-hydroxy aromatic and heterocyclic amines.
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1997) Vol. 38, No. 0, pp. 353.
Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research. San Diego, California, USA. April 12-16, 1997.
ISSN: 0197-016X.
AU Ozawa, S. [Reprint author]; Lang, N. P.; Kadlubar, F. F.
AN 1997:232563 BIOSIS

L49 ANSWER 24 OF 103 MEDLINE on STN DUPLICATE 14
TI **Phenol sulfotransferase** pharmacogenetics in humans: association of common SULT1A1 **alleles** with TS **PST** phenotype.
SO Biochemical and biophysical research communications, (1997 Oct 9) Vol. 239, No. 1, pp. 298-304.
Journal code: 0372516. ISSN: 0006-291X.
AU Raftogianis R B; Wood T C; Otterness D M; Van Loon J A; Weinshilboum R M
AN 1998005125 MEDLINE

L49 ANSWER 25 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 15
TI Metabolic differences and their impact on human disease - **Sulfotransferase** and colorectal cancer
SO ENVIRONMENTAL TOXICOLOGY AND PHARMACOLOGY, (DEC 1997) Vol. 4, No. 3-4, pp. 277-281.
ISSN: 1382-6689.
AU Frame L T; Gatlin T L; Kadlubar F F; Lang N P (Reprint)
AN 1998:103809 SCISEARCH

L49 ANSWER 26 OF 103 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI **Phenol sulfotransferase** (**PST**) molecular pharmacogenetics.
SO Clinical Pharmacology and Therapeutics, (1997) Vol. 61, No. 2, pp. 234.
Meeting Info.: Abstracts of Papers to be presented at the Ninety-eight Annual Meeting of the American Society for Clinical Pharmacology and Therapeutics. San Diego, California, USA. March 5-8, 1997.

CODEN: CLPTAT. ISSN: 0009-9236.
AU Raftogianis, R.; Wood, T.; Her, C.; Van Loon, J.; Weinshilboum, R.
AN 1997:188869 BIOSIS

L49 ANSWER 27 OF 103 MEDLINE on STN DUPLICATE 16
TI Enzymology of human cytosolic **sulfotransferases.**
SO The FASEB journal : official publication of the Federation of American Societies for Experimental Biology, (1997 Mar) Vol. 11, No. 4, pp. 206-16.
Ref: 106
Journal code: 8804484. ISSN: 0892-6638.
AU Falany C N
AN 97221563 MEDLINE

L49 ANSWER 28 OF 103 MEDLINE on STN DUPLICATE 17
TI High level expression and characterization of recombinant human hippocampus **phenol sulfotransferase**: a novel **phenol**-sulfating form of **phenol sulfotransferase**
.
SO Protein expression and purification, (1997 Oct) Vol. 11, No. 1, pp. 125-34.
Journal code: 9101496. ISSN: 1046-5928.
AU Hwang S R; Palkovits M; Hook V Y
AN 97469631 MEDLINE

L49 ANSWER 29 OF 103 MEDLINE on STN DUPLICATE 18
TI Sulfation and **sulfotransferases** 1: **Sulfotransferase** molecular biology: cDNAs and genes.
SO The FASEB journal : official publication of the Federation of American Societies for Experimental Biology, (1997 Jan) Vol. 11, No. 1, pp. 3-14.
Ref: 99
Journal code: 8804484. ISSN: 0892-6638.
AU Weinshilboum R M; Otterness D M; Aksoy I A; Wood T C; Her C; Raftogianis R B
AN 97186544 MEDLINE

L49 ANSWER 30 OF 103 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 19
TI Pentachlorophenol enhances 9-hydroxybenzo[a]pyrene-induced hepatic DNA adduct formation in vivo and inhibits microsomal epoxide hydrolase and glutathione S-transferase activities in vitro. Likely inhibition of epoxide detoxication by pentachlorophenol
SO Archives of Toxicology (1996), 70(11), 696-703
CODEN: ARTODN; ISSN: 0340-5761
AU Moorthy, Bhagavatula; Randerath, Kurt
AN 1996:664222 HCAPLUS
DN 126:2905

L49 ANSWER 31 OF 103 MEDLINE on STN DUPLICATE 20
TI cDNA cloning and expression of a new form of human aryl **sulfotransferase.**
SO The international journal of biochemistry & cell biology, (1996 May) Vol. 28, No. 5, pp. 565-71.
Journal code: 9508482. ISSN: 1357-2725.
AU Zhu X; Veronese M E; Iocco P; McManus M E
AN 96252882 MEDLINE

L49 ANSWER 32 OF 103 MEDLINE on STN DUPLICATE 21
TI Human **phenol sulfotransferase** pharmacogenetics: STP1 gene cloning and structural characterization.
SO Pharmacogenetics, (1996 Dec) Vol. 6, No. 6, pp. 473-87.
Journal code: 9211735. ISSN: 0960-314X.
AU Raftogianis R B; Her C; Weinshilboum R M
AN 97166466 MEDLINE

L49 ANSWER 33 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Human dehydroepiandrosterone **sulfotransferase** pharmacogenetics: Quantitative western analysis and gene sequence **polymorphisms**
SO JOURNAL OF STEROID BIOCHEMISTRY AND MOLECULAR BIOLOGY, (DEC 1996) Vol. 59, No. 5-6, pp. 467-478.
ISSN: 0960-0760.
AU Wood T C (Reprint); Her C; Aksoy I; Otterness D M; Weinshilboum R M
AN 1997:97336 SCISEARCH

L49 ANSWER 34 OF 103 MEDLINE on STN
TI Human **phenol sulfotransferase** STP2 gene: molecular cloning, structural characterization, and chromosomal localization.
SO Genomics, (1996 May 1) Vol. 33, No. 3, pp. 409-20.
Journal code: 8800135. ISSN: 0888-7543.
AU Her C; Raftogianis R; Weinshilboum R M
AN 96299636 MEDLINE

L49 ANSWER 35 OF 103 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Human **sulfotransferase** pharmacogenetics: STP2 gene, structural characterization and chromosomal localization.
SO Clinical Pharmacology and Therapeutics, (1996) Vol. 59, No. 2, pp. 216.
Meeting Info.: Ninety-seventh Annual Meeting of the American Society for Clinical Pharmacology and Therapeutics. Lake Buena Vista, Florida, USA. March 20-22, 1996.
CODEN: CLPTAT. ISSN: 0009-9236.
AU Her, Chengtao; Raftogianis, Rebecca; Weinshilboum, Richard
AN 1996:204047 BIOSIS

L49 ANSWER 36 OF 103 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 22
TI Detection of a SacI restriction fragment length **polymorphism** at the human **phenolsulphotransferase** locus.
SO Clinical Genetics, (1996) Vol. 49, No. 3, pp. 164-165. .
ISSN: 0009-9163 CODEN: CLGNAY
AU Jones A.L.; Roberts R.C.; Coughtrie M.W.H.
AN 96154087 EMBASE

L49 ANSWER 37 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Site-directed **mutagenesis** of rat hepatic hydroxysteroid **sulfotransferases**
SO BIOCHIMICA ET BIOPHYSICA ACTA-PROTEIN STRUCTURE AND MOLECULAR ENZYMOLOGY, (5 SEP 1996) Vol. 1296, No. 2, pp. 159-166.
ISSN: 0167-4838.
AU Homma H (Reprint); Ogawa K; Hirono K; Morioka Y; Hirota M; Tanahashi I; Matsui M
AN 1996:673362 SCISEARCH

L49 ANSWER 38 OF 103 MEDLINE on STN DUPLICATE 23
TI Genomic organization and DNA sequences of two human **phenol sulfotransferase** genes (STP1 and STP2) on the short arm of chromosome 16.
SO Biochemical and biophysical research communications, (1996 Nov 1) Vol. 228, No. 1, pp. 134-40.
Journal code: 0372516. ISSN: 0006-291X.
AU Dooley T P; Huang Z
AN 97069665 MEDLINE

L49 ANSWER 39 OF 103 MEDLINE on STN DUPLICATE 24
TI Phenotypic variation in xenobiotic metabolism and adverse environmental response: focus on sulfur-dependent detoxification pathways.
SO Toxicology, (1996 Jul 17) Vol. 111, No. 1-3, pp. 43-65. Ref: 145
Journal code: 0361055. ISSN: 0300-483X.
AU McFadden S A
AN 96319826 MEDLINE

L49 ANSWER 40 OF 103 MEDLINE on STN DUPLICATE 25
TI Proposed active site domain in estrogen **sulfotransferase** as determined by **mutational** analysis.
SO Proceedings of the National Academy of Sciences of the United States of America, (1995 Dec 19) Vol. 92, No. 26, pp. 12328-32.
Journal code: 7505876. ISSN: 0027-8424.
AU Driscoll W J; Komatsu K; Strott C A
AN 96109259 MEDLINE

L49 ANSWER 41 OF 103 MEDLINE on STN DUPLICATE 26
TI Human platelet **phenolsulfotransferases**: cDNA cloning, stable expression in V79 cells and identification of a novel **allelic variant** of the **phenol**-sulfating form.
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=> save temp l49 sult1a1/a
ANSWER SET L49 HAS BEEN SAVED AS 'SULT1A1/A'

=> d ab 8-10,15-17,21-29,32,36,38,39,41,52,58,67,69,75,77,79,92,95

L49 ANSWER 8 OF 103 MEDLINE on STN DUPLICATE 5
AB Three related forms of **phenol sulfotransferase** (PSULT), thermostable ST1A2 (SULT1A2hum) and ST1A3 (SULT1A1hum) and a thermolabile TL-**PST** (SULT1A3hum), are known to exist in human livers. Thermostable forms, whose activities are **polymorphically** distributed, have been shown to mediate the bioactivation of carcinogenic N-hydroxy arylamines and heterocyclic amines. To clarify the nature of the sulfation **polymorphism**, the study compared the expressed levels of ST1A2, ST1A3 and TL-**PST** mRNAs in human livers by the method of reverse-transcriptase polymerase chain reaction (RT-PCR), utilizing HindIII, BamHI and SnaBI sites which were unique to the above PSULT cDNAs, respectively. Of the PCR products derived from human liver (n = 26), 43-89, < 1-29 and < 1-21% showed the restriction pattern characteristic for ST1A3, ST1A2 and TL-**PST** cDNAs, respectively, thus indicating that ST1A3 mRNA is the major transcript. Hepatic p-nitrophenol and dopamine sulfation rates ranged from 440-2670 and < 5-460 pmol/min per mg protein in the 26 individuals, respectively. The observed differences in the ST1A3 and TL-**PST** mRNA levels were consistent with the differences in p-nitrophenol and dopamine sulfations. Relative levels of hepatic ST1A3 mRNA were non-normally distributed and correlated significantly with p-nitrophenol sulfation. In addition, **variant** forms of ST1A3 mRNA encoding Arg213His and Met223Val were detected in human livers. With regard to Arg213His, 28 individuals who had homozygous 213Arg **alleles**, 15 individuals who were heterozygotes and nine homozygous 213His individuals were found by a newly established genotyping method among 52 human liver samples. Frequency of 223Val **allele** was apparently lower than that of 213His **allele**, as no homozygous 223Val individual and only three individuals who were heterozygotes (223Met/Val) were observed among 52 individuals. These results suggest that regulation of p-nitrophenol sulfation occurs at the level of gene transcription of ST1A3 which is the major transcript of the three PSULT mRNAs and that a polygenic basis for the apparent genetic **polymorphism** of sulfation was likely because of the existence of ST1A3 **variants.**

L49 ANSWER 9 OF 103 MEDLINE on STN DUPLICATE 6
AB Cytosolic **phenol sulfotransferases** (**PST**) catalyze the sulfation/sulfonation of various **phenolic** agents, including catecholamines, thyroid hormones, and drugs (e.g., minoxidil and acetaminophen), which usually results in the inactivation and subsequent excretion of the compound. Our recent efforts have focused on the cloning

and sequencing of the human gene family encoding the **PST** isozymes, and the results are summarized in this article. Multiple **PST** cDNA isolates have been cloned in various laboratories representing **alleles** of three **phenol sulfotransferase** gene loci termed as STP1, STP2, and STM. All three genes have been mapped precisely to a small region on human chromosome 16p12.1-p11.2 (homologous to mouse chromosome 7). The two most closely related genes, STP1 and STP2, encode isozymes of **phenol** -preferring **PST** (P-**PST**) and have been mapped to a single genomic cosmid clone, thus in proximity to one another. The STM gene encoding the monoamine neurotransmitter-preferring **PST** (M-**PST**) exhibits a lower level of similarity relative to STP1 and STP2. Genomic clones have been sequenced to determine the genomic organization for each of the three highly related genes. All contain seven coding exons, with conserved intron-exon boundaries. Sequencing of individual cDNA isolates from various tissues has revealed heterogeneity in the 5' nontranslated regions, likely due to tissue-specific promoter utilization (or perhaps alternative splicing). DNA and protein **polymorphisms** have been identified in the population and may be useful for molecular genetic studies of the variability in the metabolism of catecholamines, thyroid hormones, and **phenolic** drugs, and possibly neuropsychiatric or other metabolic disorders.

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AB Sulfation is a common final step in the biotransformation of xenobiotics and is traditionally associated with inactivation. However, the sulfate group is electron-withdrawing and may be cleaved off heterolytically in some molecules leading to electrophilic cations which may form adducts with DNA and other important cellular structures. Since endogenous **sulfotransferases** do not appear to be expressed in indicator cells of standard **mutagenicity** tests, rat and human **sulfotransferases** have been stably expressed in his(-) Salmonella typhimurium strain TA1538 and Chinese hamster V79 cells. Using these recombinant indicator cells, **sulfotransferase**-dependent genotoxic activities were detected with N-hydroxy-2-acetylaminofluorene, 2-acetylaminofluorene (in the presence of co-expressed rat cytochrome P450 1A2), hycanthone, 1'-hydroxysafrole, alpha-hydroxytamoxifen and various benzylic alcohols derived from polycyclic aromatic hydrocarbons. In several cases, it was critical that the reactive sulfuric acid conjugates were formed directly within the indicator cells, owing to the inefficient penetration of cell membranes. In other eases, spontaneous benzylic substitution reactions with medium components, such as halogenide ions or amino acids, led to secondary, membrane-penetrating reactive species. Different **sulfotransferases**, including related forms from rat and human, substantially differed in their substrate specificity towards the investigated promutagens. It is known that some **sulfotransferases** are expressed with high tissue and cell type specificities, This site-dependent expression together with the limitations in the distribution of reactive sulfuric acid conjugates may explain organotropic effects of compounds activated by this metabolic pathway. (C) 1998 Elsevier Science Ireland Ltd. All rights reserved.

L49 ANSWER 15 OF 103 MEDLINE on STN DUPLICATE 10

AB It has become clear that several **polymorphisms** of human drug-metabolizing enzymes influence an individual's susceptibility for chemical carcinogenesis. This review gives an overview on relevant **polymorphisms** of four families of drug-metabolizing enzymes. Rapid acetylators (with respect to N-acetyltransferase NAT2) were shown to have an increased risk of colon cancer, but a decreased risk of bladder cancer. In addition an association between a NAT1 **variant allele** (NAT*10, due to **mutations** in the polyadenylation site causing approximately two fold higher activity) and colorectal cancer among NAT2 rapid acetylators was observed, suggesting a possible interaction between NAT1 and NAT2. Glutathione S-transferases M1 and T1

(GSTM1 and GSTT1) are **polymorphic** due to large deletions in the structural gene. Meta-analysis of 12 case-control studies demonstrated a significant association between the homozygous deletion of GSTM1 (GSTM1-0) and lung cancer (odds ratio: 1.41; 95% CI: 1.23-1.61). Combination of GSTM1-0 with two **allelic variants** of cytochrome P4501A1 (CYP1A1), CYP1A1 m2/m2 and CYP1A1 Val/Val further increases the risk for lung cancer. Indirect mechanisms by which deletion of GSTM1 increases risk for lung cancer may include GSTM1-0 associated decreased expression of GST M3 and increased activity of CYP1A1 and 1A2. Combination of GST M1-0 and NAT2 slow acetylation was associated with markedly increased risk for lung cancer (odds ratio: 7.8; 95% CI: 1.4-78.7). In addition GSTM1-0 is clearly associated with bladder cancer and possibly also with colorectal, hepatocellular, gastric, esophageal (interaction with CYP1A1), head and neck as well as cutaneous cancer. In individuals with the GSTT1-0 genotype more chromosomal aberrations and sister chromatid exchanges (SCEs) were observed after exposure to 1,3-butadiene or various haloalkanes or haloalkenes. Evidence for an association between GSTT1-0 and myelodysplastic syndrome and acute lymphoblastic leukemia has been presented. A **polymorphic** site of GSTP1 (valine to isoleucine at codon 104) decreases activity to several carcinogenic diol epoxides and was associated with testicular, bladder and lung cancer. Microsomal expoxide hydrolase (mEH) is **polymorphic** due to amino acid variation at residues 113 and 139. **Polymorphic variants** of mEH were associated with hepatocellular cancer (His-113 **allele**), ovarian cancer (Tyr-113 **allele**) and chronic obstructive pulmonary disease (His-113 **allele**). Three human **sulfotransferases** (STs) are regulated by genetic **polymorphisms** (hDHEAST, hM-**PST**, TS **PST**). Since a large number of environmental **mutagens** are activated by STs an association with human cancer risk might be expected.

L49 ANSWER 16 OF 103 MEDLINE on STN DUPLICATE 11

AB During studies designed to subclone human **phenol sulfotransferase** (STP and STM) sequences for use in heterologous E. coli-based expression systems, we designed two oligonucleotide primers that would allow for the simultaneous PCR amplification of expression cassettes containing the coding regions of the STP1, STP2 and STM cDNAs. Following total RNA isolation from human liver, reverse transcription of cDNA, PCR amplification under standard conditions, plasmid subcloning and restriction analysis to select for suitable ST recombinants, we recovered plasmids containing inserts corresponding to STP1, STP2 and STM. However, ten additional, closely related but apparently novel ST sequences were also isolated. Alignments of the three known ST sequences (and one published **allelic variant**) with these new clones revealed that each one appears to be a PCR-generated modular chimera possessing a combination of DNA segments derived from STP1, STP2 and STM. This observation should serve as an alert to the potential pitfalls of using PCR techniques for the cloning of highly related genes and their cDNA products, especially when PCR primer design allows for the amplification of multiple products in a single reaction.

L49 ANSWER 17 OF 103 MEDLINE on STN DUPLICATE 12

AB **Phenol sulfotransferases** (**PST**) catalyze the sulfonation of catecholamines, thyroid hormones and **phenolic** drugs. At least two major forms of human **PST** enzyme have been characterized biochemically from liver, platelets and other tissues, the **phenol**-preferring **PST** (P-**PST**) and the monoamine neurotransmitter-preferring **PST** (M-**PST**). Molecular cloning efforts worldwide over the past 7 years have resulted in the identification of numerous **PST** cDNA isolates representing **alleles** of three human **PST** gene loci termed as STP1, STP2 and STM. All three genes have been mapped precisely to a small region on human chromosome 16p12.1-p11.2 (homologous to mouse chromosome 7), using somatic cell hybrids and cosmid clones. The two most closely related genes, STP1 and STP2, encoding P-**PST** isozymes have been

mapped to a single cosmid clone and are, therefore, in close proximity to one another. STP1 and STP2 are approximately 96% identical at the amino acid sequence level, whereas, the STM gene (encoding M-**PST**) exhibits a lower level of identity (approximately 93-90.5%) relative to STP1 and STP2. STM is located at a distance of ca. 100 Kb from the STP1 and STP2 doublet. One may speculate that the three genes arose by gene duplication and/or gene conversion in humans. Genomic clones have been sequenced to determine the genomic organization for each of the three highly-related genes. All contain seven coding exons, with conserved intron exon boundaries. Sequencing of individual cDNA isolates of STP1 and STM from various tissues has revealed significant heterogeneity in the 5' nontranslated region, likely due to alternative splicing and/or tissue-specific promoter utilization. DNA **polymorphisms** have been detected in these genes in the human population and may be useful for molecular genetic studies of the metabolism of endogenous and xenobiotic **phenolic** molecules. Recent advances in the molecular biology of the human **PST** gene family are summarized.

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AB Glucuronidation and sulfation are phase 2 metabolic reactions catalyzed by large families of different isoenzymes in man. The textbook view that glucuronidation and sulfation lead to the production of harmless conjugates for simple excretion is not valid. Biologically active and toxic sulfates and glucuronides are produced and lead to adverse drug reactions, including immune hypersensitivity. Considerable variation in xenobiotic conjugation is observed as a result of altered expression of UDP-glucuronosyltransferases (UGTs) and **sulfotransferases** (STs). Recent cloning and expression of human cDNA encoding UGTs and STs has facilitated characterization of isoform substrate specificity, which has been further validated using specific antibodies and human tissue fractions. The availability of cloned/expressed human enzymes and specific antibodies has enabled the investigation of xenobiotic induction and metabolic disruption leading to adverse responses. Genetic **polymorphisms** of glucuronidation and sulfation are known to exist although the characterization and assessment of the importance of these variations are hampered by appropriate ethical studies in man with suitable safe model compounds. Genetic analysis has allowed molecular identification of defects in well-known hyperbilirubinemias. However, full characterization of the specific functional roles of human UGTs and STs requires rigorous kinetic and molecular analyses of the role of each enzyme in vivo through the use of specific antibodies and inhibitors. This will lead to the better prediction of variation of xenobiotic glucuronidation and sulfation in man.

L49 ANSWER 22 OF 103 MEDLINE on STN DUPLICATE 13

AB Sulfation is an important conjugation reaction in the metabolism of a diversity of xenobiotics and endogenous compounds such as drugs, food additives, carcinogens, steroids and neurotransmitters. Sulfate conjugation is catalyzed by various kinds of **sulfotransferase** (ST) such as hydroxysteroid ST (HS-ST), **phenol** ST (P-ST) and estrogen ST (E-ST) present in cytosols. Our laboratory has been studying the multiplicity of rat and mouse STs. We found that tertiary amines such as triethylamine selectively inhibited rat hepatic HS-ST. Developmental changes and zonal distributions of rat liver HS-ST and P-ST isoenzymes provided evidence for their complex regulatory mechanisms. Studies on site-directed **mutagenesis** and chimeras of rat HS-ST cDNAs, ST-40 and ST-20, revealed the importance of the C-terminal region for the substrate specificity and involvement of multiple regions for the enzyme activities. These two cDNAs have been mapped to the same chromosomal region 1q21.3-->q22.1 by fluorescence in situ hybridization. Mouse olfactory P-ST was present in the cytoplasm of olfactory sustentacular cells and its cloning study revealed that it is 94% identical with rat ST1C1 in amino acid sequences.

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L49 ANSWER 24 OF 103 MEDLINE on STN DUPLICATE 14

AB The **phenol sulfotransferases** (PSTs) catalyze the sulfation of both small planar **phenols** and **phenolic** monoamines. Three highly homologous **PST** genes, SULT1A1, SULT1A2, and SULT1A3, are known to exist in humans. The prototypic biochemical phenotype associated with the enzyme encoded by SULT1A1 is the thermal stable (TS) sulfation of 4 microM 4-nitrophenol (TS **PST** activity). Biochemical pharmacogenetic studies have demonstrated that individual variation in both TS **PST** activity and thermal stability in humans are inherited. As a step toward understanding molecular mechanisms responsible for the genetic regulation of PSTs in humans, we report here common SULT1A1 nucleotide **polymorphisms** that are associated with phenotypic variation in both platelet TS **PST** activity and thermal stability. When 905 human subjects were phenotyped for platelet TS **PST** activity and thermal stability, activity varied more than 50-fold, and thermal stability varied over 10-fold. DNA was isolated from the blood of 33 of these subjects selected on the basis of "extreme" TS **PST** phenotypes: high activity and high thermal stability; low activity and low thermal stability; or low activity and high thermal stability. These 33 subjects were genotyped for SULT1A1 by PCR amplification and sequencing of the entire open reading frame (ORF) as well as approximately 1 kb of intron DNA sequence. One common **allele**, SULT1A1*2, was uniformly associated with both very low TS **PST** activity and low thermal stability. The **allele** frequency of SULT1A1*2 in a randomly selected population sample of 150 Caucasian blood donors was 0.31 (31%), indicating that approximately 9% of this population would be homozygous for that **allele.**

L49 ANSWER 25 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 15

AB Gene-environment interaction is an important aspect of human cancer risk. Genetic **polymorphisms** in acetylation and N-oxidation have previously been described regarding their impact on the heterocyclic amine-induced risk for colon cancer. Here, we report that another enzyme involved in the metabolism of food-borne carcinogens, **sulfotransferase** (ST1A3 measured by 2-naphthol activity), may function as a potential protective factor for colon cancer in humans. Initially characterized in human liver and colon (Chou et al., 1995), TS-**PST** activity can also be measured in platelets. A simple microtiter-based colorimetric technique was developed for use in this case-control study. African-Americans had a higher mean ST activity than Caucasians (2.32 +/- 0.24 versus 1.77 +/- 0.09 nmols/min per mg cytosolic protein, P = 0.036). Furthermore, the slow ST phenotype (ST less than or equal to 1.53) was more frequently associated with colon cancer than controls (57 versus 40%, P = 0.026). These data suggest that the ST1A3 isoform may play a role in the differential risk for colorectal cancer. (C) 1997 Elsevier Science B.V.

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L49 ANSWER 27 OF 103 MEDLINE on STN DUPLICATE 16

AB Conjugation of many xenobiotics, drugs, and endogenous compounds with a sulfonate moiety is an important reaction in their biotransformation. Sulfation of these compounds generally results in a decrease in biological activity and an increase in their urinary excretion. However, in certain instances, sulfation results in bioactivation to reactive electrophilic or therapeutically active forms. At least four cytosolic **sulfotransferases** (STs) have been identified and characterized from human tissues. These enzymes are two forms of **phenol** ST (**PST**), the **phenol**-sulfating and the monoamine-sulfating

forms of **PST** (P-**PST** and M-**PST**, respectively), an estrogen **sulfotransferase** (EST), and a hydroxysteroid ST, dehydroepiandrosterone ST (DHEA-ST). Although four cytosolic STs have been well characterized in human tissues, evidence is accumulating for the presence of **allelic** forms or additional distinct forms of the STs in human tissues. The STs possess distinct but overlapping substrate specificities, and all of the STs are capable of conjugating both xenobiotic and endogenous compounds. The individual forms of ST may display distinct patterns of tissue specific expression and different mechanisms of regulation. Although the role of sulfation in drug metabolism is well recognized, an increased understanding of the biochemistry and molecular biology of the STs should also provide additional information as to their functions in many normal physiologic processes.

L49 ANSWER 28 OF 103 MEDLINE on STN DUPLICATE 17

AB **Phenol sulfotransferases** (PSTs) represent a family of **sulfotransferase** enzymes that modify the biologic activities and excretion of **phenolic** compounds and monoamines. A novel human hippocampal **PST** (H-**PST**) cDNA with homology to **phenol** (P) and monoamine (M) forms of **PST** was previously isolated from brain. To compare the biochemical properties of H-**PST** with that of **phenol** (P-**PST**) and monoamine (M-**PST**) **sulfotransferases**, high level expression of recombinant H-**PST** was achieved in this study with the pET3c vector in BL21(DE3) Escherichia coli cells. Expression was demonstrated by isopropyl beta-D-thiogalactopyranoside induction of 34-kDa H-**PST** that represented 5-10% of total E. coli proteins. Purification by ion-exchange chromatography on DEAE-Sepharose yielded more than 2 mg of H-**PST**. Characterization showed that H-**PST** exists as a homodimer of 60-65 kDa by gel filtration chromatography. H-**PST** prefers p-nitrophenol as substrate and does not sulfate dopamine or neuropeptide substrates. Kinetic studies showed that H-**PST** possessed K(m(app)) and Vmax(app) values of 3 microM p-nitrophenol and 160 nmol/min/mg, respectively. H-**PST** was sensitive to inhibition by DCNP (2,6-dichloro-4-nitrophenol). H-**PST** is thermolabile since its activity was reduced upon preincubation at 37 degrees C. These results indicate that H-**PST** shows similarities and differences compared to P-**PST** and M-**PST sulfotransferases.** P-**PST** prefers p-nitrophenol as substrate, is sensitive to inhibition by DCNP, and is thermostable; in contrast, M-**PST** prefers monoamines as substrate, is not sensitive to DCNP, and is thermolabile. The distinct profile of biochemical properties of H-**PST**, and its primary sequence homology to P-**PST** and M-**PST**, suggests that H-**PST** represents a novel **allelic variant** of human **phenol sulfotransferases.** Importantly, this study demonstrates that high level expression of H-**PST** allows determination of distinguishing characteristics of **variant** forms of PSTs.

L49 ANSWER 29 OF 103 MEDLINE on STN DUPLICATE 18

AB **Sulfotransferase** (ST) enzymes catalyze the sulfate conjugation of many hormones, neurotransmitters, drugs, and xenobiotic compounds. These reactions result in enhanced renal excretion of the sulfate-conjugated reaction products, but they can also lead to the formation of "bioactivated" metabolites. ST enzymes are members of an emerging gene superfamily that presently includes **phenol** ST (**PST**), hydroxysteroid ST (HSST), and, in plants, flavonol ST (FST) "families," members of which share at least 45% amino acid sequence identity. These families can be further subdivided into "subfamilies" that are at least 60% identical in amino acid sequence. For example, the **PST** family includes both **PST** and estrogen ST (EST) subfamilies. Amino acid sequence motifs exist within ST enzymes that are conserved throughout phylogeny. These signature sequences may be involved

in the binding of 3'-phosphoadenosine-5 '-phosphosulfate, the cosubstrate for the sulfonation reaction. There are presently five known human cytosolic ST enzymes: an EST, an HSST, and three PSTs. cDNAs and genes for all of these enzymes have been cloned, and chromosomal localizations have been reported for all five genes. Genes for these human enzymes, as well as those of other mammalian cytosolic ST enzymes that have been cloned, show a high degree of structural homology, with conservation of the locations of most intron/exon splice junctions. Human ST enzyme expression varies among individuals. Functionally significant genetic **polymorphisms** for ST enzymes in humans have been reported, and other molecular genetic mechanisms that might be involved in the regulation of the expression of these enzymes are being explored. Knowledge of the molecular biology of cytosolic ST enzymes, when placed within a context provided by decades of biochemical research, promises to significantly enhance our understanding of the regulation of the sulfate conjugation of hormones, neurotransmitters, and drugs.

L49 ANSWER 32 OF 103 MEDLINE on STN DUPLICATE 21
AB Sulfate conjugation catalysed by **phenol sulfotransferase** (**PST**) is an important pathway in the metabolism of many drugs. Two isoforms of **PST** have been characterized biochemically in human tissues-a thermostable (TS), or **phenol**-metabolizing (P) and a thermolabile (TL), or monoamine-metabolizing (M) form. Pharmacogenetic studies of TS and TL **PST** activities in the human blood platelet showed that the activities of these two isoforms were regulated by separate genetic **polymorphisms.** Subsequently, a series of TS **PST** cDNAs were cloned, and, based on sequence homology, those cDNAs could be classified as members of two separate subgroups, designated here as 'TS PST1' and 'TS PST2'-indicating the existence of three rather than two **PST** isoforms; TS PST1, TS PST2 and TL **PST.** The genes encoding TS PST2, STP2, and TL **PST**, STM, have been cloned, structurally characterized and mapped to chromosome 16-the same chromosome on which the TS PST1 gene, STP1, is localized. As a step toward molecular pharmacogenetic studies of sulfate conjugation in humans, we set out to clone and structurally characterize STP1, the remaining uncharacterized human **PST** gene. We found that STP1 spanned approximately 4.4 kb and contained 9 exons. The first two exons, IA and IB, were identified by performing 5'-rapid amplification of cDNA ends (RACE) with human liver cDNA as template. Exons IA and IB were noncoding and represented two different cDNA 5'-untranslated region sequences. No canonical TATA box sequences were present within the 5'-flanking regions of the gene, i.e. regions flanking exons IA and IB. Finally, use of the long polymerase chain reaction made it possible to determine that STP1 is located approximately 45 kb 5'-upstream from STP2 on the short arm of human chromosome 16. Cloning and structural characterization of STP1, when combined with knowledge of the structures of STP2 and STM, will make it possible to study the molecular basis for the genetic regulation of **PST** activity in human tissue.

L49 ANSWER 36 OF 103 EMBASE DUPLICATE 22

L49 ANSWER 38 OF 103 MEDLINE on STN DUPLICATE 23
AB A family of human **phenol sulfotransferase** genes has been suggested by the cloning of numerous cDNA isolates from different tissues. We have previously cloned and sequenced the STM gene encoding the monoamine neurotransmitter-preferring **sulfotransferase**, M-**PST**, and a portion of the STP1 gene encoding the **phenol** -preferring isozyme, P-PST1 (BBRC 205, 1325-1332; Genomics 18, 440-443). Both genes were mapped to a small region on the short arm of chromosome 16 (BBRC 205, 482-489). Here we report on the sequencing and genomic organization of the STP1 and STP2 genes from a single cosmid clone obtained from chromosome 16p12.1-p11.2. STP1 and STP2 are 95.9% identical at the amino acid sequence level, whereas the STM gene is only 92.9% and 90.5% identical to STP1 and STP2, respectively. Alignment of the genomic

sequences indicated that all three genes have 7 coding exons and conserved intron-exon boundaries. These results facilitated the assignment of previously published cDNA isolates as "**alleles**" of the individual STM, STP1, and STP2 loci on 16p, and provide to us a greater understanding of the complexity and roles of the **phenol sulfotransferase** gene family in the metabolism of endogenous and xenobiotic agents.

L49 ANSWER 39 OF 103 MEDLINE on STN DUPLICATE 24
AB Proper bodily response to environmental toxicants presumably requires proper function of the xenobiotic (foreign chemical) detoxification pathways. Links between phenotypic variations in xenobiotic metabolism and adverse environmental response have long been sought. Metabolism of the drug S-carboxymethyl-L-cysteine (SCMC) is **polymorphous** in the population, having a bimodal distribution of metabolites, 2.5% of the general population are thought to be nonmetabolizers. The researchers developing this data feel this implies a **polymorphism** in sulfoxidation of the amino acid cysteine to sulfate. While this interpretation is somewhat controversial, these metabolic differences reflected may have significant effects. Additionally, a significant number of individuals with environmental intolerance or chronic disease have impaired sulfation of **phenolic** xenobiotics. This impairment is demonstrated with the probe drug acetaminophen and is presumably due to starvation of the **sulfotransferases** for sulfate substrate. Reduced metabolism of SCMC has been found with increased frequency in individuals with several degenerative neurological and immunological conditions and drug intolerances, including Alzheimer's disease, Parkinson's disease, motor neuron disease, rheumatoid arthritis, and delayed food sensitivity. Impaired sulfation has been found in many of these conditions, and preliminary data suggests that it may be important in multiple chemical sensitivities and diet responsive autism. In addition, impaired sulfation may be relevant to intolerance of **phenol**, tyramine, and phenylic food constituents, and it may be a factor in the success of the Feingold diet. These studies indicate the need for the development of genetic and functional tests of xenobiotic metabolism as tools for further research in epidemiology and risk assessment.

L49 ANSWER 41 OF 103 MEDLINE on STN DUPLICATE 26
AB We have isolated cDNA clones encoding **phenol**- and monoamine-sulfating **phenolsulfotransferases**, using the reverse transcription-polymerase chain reaction method with human platelet mRNA as template. These cDNAs were stably expressed in the Chinese hamster fibroblast cell line V79 and their substrate specificities towards known **sulfotransferase** isoenzyme-selective compounds determined. The nucleotide and derived amino acid sequences of the monoamine-sulfating form were identical to those previously identified in human liver and brain, but the cDNA for the human platelet **phenol**-sulfating form we isolated contained 5 and 2 amino acid changes from the two previously published sequences from human liver and brain, suggesting that we have identified a new **allelic variant** of human **phenol**-sulfating **phenolsulfotransferase.**

L49 ANSWER 52 OF 103 MEDLINE on STN DUPLICATE 32
AB The gene encoding human **phenol**-preferring **phenol sulfotransferase** (STP) has been cloned and mapped to chromosome 16p. A HindIII RFLP in this gene is described.

L49 ANSWER 58 OF 103 MEDLINE on STN DUPLICATE 35

L49 ANSWER 67 OF 103 MEDLINE on STN DUPLICATE 41
AB Human tissues contain at least three well-characterized cytoplasmic **sulfotransferase** (ST) enzymes, thermostable (TS) and thermolabile (TL) forms of ST (**PST**) and dehydroepiandrosterone (DHEA) ST. Both forms of **PST** are expressed in an easily accessible human

tissue, the blood platelet. The presence of **PST** in blood platelets made it possible to perform pharmacogenetic studies of these enzymes in humans. Those studied demonstrated that TS and TL **PST** activities in the human platelet are regulated by separate, common genetic **polymorphisms.** Furthermore, the platelet activity of TS, but not of TL **PST** is correlated with levels of this enzyme activity in other human tissues such as liver, jejunal mucosa and cerebral cortex. The pharmacogenetic strategy used to study TS and TL **PST** could not be applied to DHEA ST since that enzyme is not expressed in human blood elements. However, DHEA ST is expressed in the liver. When 94 samples of human hepatic biopsy tissue obtained during clinically-indicated surgery were studied, there was a 4.6-fold range of DHEA ST activity levels and a bimodal frequency distribution, with approximately 25% of the samples included in a 'high activity' subgroup. The presence of bimodality raised the possibility that human DHEA ST activity might also be regulated by a genetic **polymorphism.** Since a cDNA for human hepatic DHEA ST has been cloned, it will now be possible to study molecular genetic mechanisms that might be involved in the regulation of individual variation in DHEA ST activity in human hepatic tissue. Pharmacogenetic studies of ST enzymes are intended, ultimately, to determine the role of inheritance in the regulation of individual variation in the sulfate conjugation of drugs,, xenobiotics, neurotransmitters and hormones in humans.

L49 ANSWER 69 OF 103 MEDLINE on STN DUPLICATE 42
AB The cDNA for human liver **phenol**-sulfating **phenol sulfotransferase** (P-**PST**) has been cloned and the active enzyme expressed in Cos cells and bacteria. Analysis of the sequence identified two cysteine residues, one of which is highly conserved in the **phenol sulfotransferase** gene family. Previous studies with the pure human liver enzyme suggested that the conserved cysteine may be involved in binding substrates. Bacterial expression of P-**PST** with the cysteine converted to a serine indicates that the cysteine is not essential for activity or substrate binding, however, the **mutant** enzyme is significantly more sensitive to thermal inactivation.

L49 ANSWER 75 OF 103 HCAPLUS COPYRIGHT 2006 ACS on STN
AB The aryl(**phenol**) **sulfotransferase** cDNAs of human and rat were cloned and sequenced. Three rat aryl(**phenol**) **sulfotransferase** clones showed genetic **polymorphism.** A human aryl(**phenol**) **sulfotransferase** clone and a rat clone showed 77% homol.

L49 ANSWER 77 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
AB We have examined the expression of platelet **phenolsulphotransferase** (**PST**) in 60 individuals. Using an antibody which recognizes both forms of **PST** present in man (P-**PST** and M-**PST**), we determined that the **polymorphism** of platelet P-**PST** activity is determined by the level of expression of the enzyme protein. The implications for susceptibility to adverse drug reactions and chemical carcinogenesis are discussed.

L49 ANSWER 79 OF 103 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 48
AB A cDNA encoding the human liver **phenol**-sulfating form of **phenol sulfotransferase** (P-**PST**) has been isolated and characterized from a λUni- Zap XR human liver cDNA library. P-**PST** is the major form of **phenol sulfotransferase** involved in drug and xenobiotic metabolism in human liver. P-**PST** is also responsible for the sulfation and activation of minoxidil to its therapeutically active sulfate ester. The full length cDNA, P-**PST**-1, is 1206 base pairs in length and encodes a 295-amino acid protein with a molecular mass of 34,097 Da. The

translation sequence of P-**PST**-1 is 96% similar to the amino acid sequences of five peptides derived from the purified protein. In vitro transcription and translation of P-**PST**-1 generated a protein that comigrates with immunoreactive P-**PST** from human liver. Significant increases in **sulfotransferase** activity toward two P-**PST**-specific substrates, minoxidil and 4-nitrophenol, were detected in cytosol prepared from COS-7 cells transfected with P-**PST**-1 in the expression vector p-SV- SPORT-1. Northern blot analysis of human liver RNA detected a transcript of approximately 1300 nucleotides in length. Characterization of P-**PST** at the molecular level provides insight into the structure and heterogeneity of this major class of drug-metabolizing enzymes.

L49 ANSWER 92 OF 103 MEDLINE on STN DUPLICATE 53

AB Platelet TS **PST** basal activity and thermal stability were measured in blood samples from 237 individuals in 50 nuclear families. Significant correlations were found among first degree relatives, confirming the previously reported familial aggregation of TS **PST** basal activity and thermal stability. Commingling analysis of basal TS **PST** activity provided evidence for multiple component distributions, and after transformation to remove skewness, segregation analysis supported a major gene hypothesis. For TS **PST** thermal stability, commingling analysis also provided evidence for multiple component distributions. However, segregation analyses were equivocal with regard to the presence of a major gene for thermal stability, since support for a major gene model depended on skewness. Bivariate commingling analysis, which examined thermal stability by simultaneously considering basal activity and activity after heating, suggested that genotypes, as defined by the inferred component distributions for TS **PST** activity, differ in thermal stability. A three-**allele** model is proposed as one hypothesis that may account for the combined results of basal activity and thermal stability. The results of this study indicate that a major gene **polymorphism** in conjunction with polygenic inheritance plays an important role in the regulation of both level of activity and thermal stability of this important drug-metabolizing enzyme in humans.

L49 ANSWER 95 OF 103 MEDLINE on STN DUPLICATE 54

AB 1. **Phenol sulfotransferase** (**PST**) catalyzes the sulfate conjugation of many **phenolic** and catechol neurotransmitters. Human tissues contain both thermostable (TS) and thermolabile (TL) forms of **PST** that differ in their substrate specificities, inhibitor sensitivities, physical properties, and regulation. 2. Individual variations in the levels of activity of both TS and TL **PST** in the human platelet are strongly influenced by inheritance. 3. Individual differences in the level of platelet TS **PST** activity are correlated with individual variations in the activity of this form of the enzyme in human cerebral cortex, liver, and intestinal mucosa. 4. There are also individual familial differences in the thermal stability of TS **PST** in the platelet. These differences are correlated with individual variations in the thermal stability of TS **PST** in cerebral cortex, liver, and intestinal mucosa. 5. Individual variations in the thermal stability of TS **PST** in hepatic tissue are associated with the presence of one or both of a pair of TS **PST** isozymes that can be separated by ion-exchange chromatography and that differ in their thermal stabilities. 6. This series of observations suggests that a structural gene **polymorphism** may be one mechanism by which inheritance controls TS **PST** in humans. The isozymes of TS **PST** in liver may represent the products of alternative **alleles** for this **polymorphism**, **alleles** that might control the structure of TS **PST** in many human tissues.

=> d ab 12,20,33-35,42,47,48,51,53,63,74,78,83,89,100

L49 ANSWER 12 OF 103 MEDLINE on STN DUPLICATE 8

AB 2-Nitropropane (2-NP) is a well-known genotoxin and carcinogen in rat liver. Several metabolic pathways, particularly cytochrome P450-, peroxidase- and **sulfotransferase**-dependent ones, have been suggested to lead to the formation of DNA-reactive species from 2-NP. Because rat liver cells express most types of xenobiotic-metabolizing enzymes, the role of specific pathways in the metabolic activation of 2-NP is difficult to assess in these cells. We have therefore investigated the genotoxicity of 2-NP and its anionic form, propane 2-nitronate (P2N), in cultured ovine seminal vesicle (OSV) cells. OSV cells lack cytochrome P450-dependent monooxygenase activity, but express prostaglandin-H-synthase (PHS) and, as we found out, **phenol sulfotransferase**. The induction of DNA repair synthesis and specific DNA modifications served as indicators for the genotoxicity of 2-NP and P2N. Both forms strongly induced repair, P2N being more active than 2-NP. The secondary nitroalkanes nitrocyclopentane and nitrocyclohexane also induced repair, whereas 1-nitropropane and the reduction product of 2-NP, acetone oxime, did not. P2N also elicited the formation of the characteristic DNA modifications 'DX1' and 8-aminodeoxyguanosine and increased the level of 8-oxodeoxyguanosine residues in the DNA. Pretreatment of OSV cells with indomethacin, an inhibitor of PHS, affected neither the induction of repair nor the formation of the DNA modifications, and P2N was not a reducing substrate for the PHS-peroxidase activity. In contrast, the **sulfotransferase** inhibitor pentachlorophenol strongly reduced genotoxicity. The results show that cytochrome P450-dependent monooxygenases are not required for the metabolic conversion of secondary nitroalkanes or their nitronates into DNA-damaging products, nor is PHS involved in the metabolic activation. Instead, the data corroborate an essential role of **sulfotransferase**(s) in the genotoxicity and carcinogenicity of secondary nitroalkanes. Moreover, it is demonstrated for the first time that these compounds can be genotoxic in cells other than hepatocytes or hepatoma cells. This implies that in species other than the rat, organs other than the liver can be targets for the genotoxicity, and possibly carcinogenicity, of secondary nitroalkanes.

L49 ANSWER 20 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

AB Activity of both the M-and P-forms of sulphotransferase (ST) was measured in platelets from patients with migraine, tension headache and controls. Mean **PST** values were 0.065+/-0.023 and 0.057+/-0.052 nmol/mg protein/min for migraine patients with and without aura. The corresponding values for tension headache and controls were 0.122+/-0.059 and 0.127+/-0.093 nmol/mg protein/min respectively ($p<0.05$). Mean MST values were not different for any of the groups, and MST and **PST** activities measured in two patients during a migraine attack were not significantly altered from baseline levels. Mean plasma inorganic sulphate concentrations and paracetamol metabolites were not significantly different in any of the groups studied. The results suggest that **PST** activity may be a factor in the aetiology of migraine.

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AB Dehydroepiandrosterone **sulfotransferase** (DHEA ST) catalyzes the sulfation of DHEA and other hydroxysteroids. DHEA ST enzymatic activity in individual human liver biopsy samples has been shown to vary over a five-fold range, and frequency distribution histograms are bimodal, with approximately 25% of subjects included in a high activity subgroup. We set out to characterize the molecular basis for variation in human liver DHEA ST activity. The first step involved performing quantitative Western analysis of cytosol preparations from 92 human liver samples that had been phenotyped with regard to level of DHEA ST enzymatic activity. There was a highly significant correlation ($r(s) = 0.635$, $P < 0.0001$) between levels of DHEA ST activity and immunoreactive protein. We next attempted to determine whether the expression of DHEA ST might be

controlled, in part, by a genetic **polymorphism.** DNA was isolated from three ''low'' and three ''high'' DHEA ST activity liver samples. Exons and the 5'-flanking region of the DHEA ST gene (STD) were amplified for each of these samples with the polymerase chain reaction (PCR). When compared with ''wild type'' STD sequence, some of the samples contained a T --> C transition at DHEA ST cDNA nucleotide 170, located within exon 2, resulting in a Met 57 --> Thr change in amino acid. Other samples contained an A --> T transversion at nucleotide 557 within STD exon 4 that resulted in a Glu 186 --> Val change. STD exons 2 and 4 were then sequenced for DNA isolated from an additional 87 liver samples that had been phenotyped with regard to level of DHEA ST enzymatic activity. The **allele** frequency for the exon 2 **polymorphism** in these samples was 0.027, whereas that for the exon 4 **polymorphism** was 0.038, but neither **polymorphism** was systematically related to the level of enzyme activity in these samples. Transient expression in COS-1 cells of cDNA that contained the nucleotide 170 and 557 **polymorphisms**, either separately or together, resulted in decreased expression of both DHEA ST enzymatic activity and level of immunoreactive protein, but only when the nucleotide 557 **variant** was present. Identification of common genetic **polymorphisms** within STD will now make it possible to test the hypothesis that those **polymorphisms** might alter in vivo expression and/or function of this important human steroid-metabolizing enzyme.

L49 ANSWER 34 OF 103 MEDLINE on STN

AB Sulfonation is an important pathway in the biotransformation of many drugs, xenobiotics, neurotransmitters, and steroid hormones. The thermostable (TS) form of **phenol sulfotransferase** (**PST**) preferentially catalyzes the sulfonation of "simple" planar **phenols**, and levels of activity of TS **PST** in human tissues are controlled by inheritance. Two different human liver TS **PST** cDNAs have been cloned that encode proteins with amino acid sequences that are 96% identical. We have determined the structure and chromosomal localization of the gene for one of these two cDNAs, STP2, as a step toward understanding molecular genetic mechanisms involved in the regulation of this enzyme activity in humans. STP2 spans approximately 5.1 kb and contains nine exons that range in length from 74 to 347 bp. The locations of most STP2 exon-intron splice junctions are identical to those of a gene for the thermolabile form of **PST** in humans, STM; a rat **PST** gene; a human estrogen ST (EST) gene, STE; and a guinea pig EST gene. The two initial STP2 exons, IA and IB, were identified by performing 5'-rapid amplification of cDNA ends with human liver cDNA as template. Exons IA and IB are noncoding and represent two different human liver TS **PST** cDNA 5'-untranslated region sequences. The two apparent 5'-flanking regions of the STP2 gene, regions flanking exons IA and IB, contain no canonical TATA boxes, but do contain CCAAT elements. STP2 was localized to human chromosome 16 by performing the PCR with DNA from NIGMS human/rodent somatic cell hybrids as template. Structural characterization of STP2 will make it possible to begin to study molecular genetic mechanisms involved in the regulation of TS **PST** activity in human tissue.

L49 ANSWER 35 OF 103 BIOSIS on STN

L49 ANSWER 42 OF 103 HCAPLUS on STN

AB Rat and human **sulfotransferases** (STs) were expressed in his- S. typhimurium strains. These new bacterial strains detected various **mutagens** which are difficult to recognize in traditional test systems, including benzylic alcs. derived from polycyclic aromatic hydrocarbons, hycanthone and 1'-hydroxysafrole. STs were also stably expressed in V79 Chinese hamster cells, which do not express endogenous ST and are suitable for the detection of genotoxic effects. Pos. responses in these test systems were observed with various benzylic alcs., including

benzo[a]pyrene-7,8,9,10-tetrols. We demonstrate that a few reactive sulfuric acid conjugates are efficiently detected as genotoxicants only when generated directly within the indicator cell.

L49 ANSWER 47 OF 103 MEDLINE on STN DUPLICATE 29

AB Several N-hydroxy metabolites of carcinogenic arylamines and heterocyclic amines were examined as substrates for bioactivation by human liver **sulfotransferases** (STs). Among the N-hydroxy derivatives studied, N-hydroxy-2-acetylaminofluorene, N-hydroxy-2-aminofluorene, N-hydroxy-4,4'-methylene-bis(2-chloroaniline), N-hydroxy-2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine, and N-hydroxy-2-amino-6-methyldipyrido[1,2-a:3',2'-d]imidazole were each metabolically activated by 3'-phosphoadenosine-5'-phosphosulfate-dependent human liver STs. No ST-mediated DNA binding of N-hydroxy-2-amino-3-methylimidazo[4,5-f]quinoline or N-hydroxy-2-amino-3,8-dimethylimidazo[4,5-f]quinoxaline was detected under our assay conditions. In the 12 human hepatic cytosols studied, the extent of 3'-phosphoadenosine-5'-phosphosulfate-dependent DNA binding of the N-hydroxy derivatives were all significantly correlated with levels of thermostable **phenol** ST (TS-**PST**) activity but not with thermolabile **phenol** ST or dehydroepiandrosterone ST activities. The propensity of these N-hydroxy arylamines and N-hydroxy heterocyclic amines to serve as selective substrates for human TS-**PST** was further confirmed by inhibition with 2,6-dichloro-4-nitrophenol and by thermostability studies. N-hydroxy-2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine and N-hydroxy-4,4'-methylene-bis(2-chloroaniline) were also used as substrates to study ST-dependent metabolic activation in other human tissue preparations. 3'-phosphoadenosine-5'-phosphosulfate-dependent DNA binding activity was detected in human liver and colon cytosols but not in pancreas, larynx, or urinary bladder epithelial cytosols. Since the TS-**PST** appears to be expressed **polymorphically** in human populations, the finding that human TS-**PST** is capable of metabolically activating N-hydroxy metabolites of several carcinogenic arylamines and heterocyclic amines suggests that TS-**PST** may have an important role in determining interindividual susceptibility to these environmental and dietary carcinogens.

L49 ANSWER 48 OF 103 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN

AB The role of human **sulfotransferase**(s) in the bioactivation of the N-hydroxy (N-OH) metabolite of the human bladder carcinogen 4-aminobiphenyl (ABP) was investigated in vitro with human tissue cytosols. Using an enzymatic assay consisting of a PAPS-regenerating system, .sup.3H N-OH-ABP, calf thymus DNA and tissue cytosols, the **sulfotransferase**-mediated metabolic activation of N-OH-ABP was determined as the PAPS-dependent covalent binding of the N-OH substrate to DNA. With cytosols prepared from various tissues, we found that the **sulfotransferase**(s) in human liver, and to a lesser extent colon, can readily metabolize N-OH-ABP, No PAPS-dependent metabolic activation was detected with cytosols prepared from human pancreas or from the carcinogen target tissue, the urinary bladder epithelium. The N-OH-ABP **sulfotransferase** activities of liver and colon cytosols from different individuals were highly correlated with their thermostable **phenol sulfotransferase** (TS-**PST**) activity (liver, $r = 0.99$, $P < 0.01$; colon, $r = 0.88$, $P < 0.01$), but not with activities for the thermolabile **phenol sulfotransferase** (TL-**PST**; liver, $r = 0.29$; colon, $r = 0.53$), or for the dehydroepiandrosterone **sulfotransferase** (DHEA-ST; liver, $r = 0.32$; colon, negligible activity), N-OH-ABP **sulfotransferase** activity was highly sensitive to inhibition by a selective TS-**PST** inhibitor, 2,6-dichloro-4-nitrophenol (IC.sub.5.sub.0 = 0.7 μM), and by p-nitrophenol, but was unaffected by competitive inhibitors of TL-**PST** (dopamine) or DHEA-ST (DHEA, DHEA-sulfate). The N-OH-ABP **sulfotransferase** activity also exhibited thermostability properties similar to that of the TS-**PST**. From these data, we

conclude that human liver TS-**PST** but not TL-**PST** or DHEA-ST can metabolically activate the proximate human carcinogen N-OH-ABP to a reactive sulfuric acid ester intermediate that binds covalently to DNA. In addition, in view of the putative role of N-OH-ABP as a major transport form of the carcinogen to the urinary bladder and of the absence of **sulfotransferase** activity in this tissue, we hypothesize that **sulfotransferase** activation in the liver may actually decrease the bioavailability of N-OH-ABP toward extrahepatic tissues and thus serve as an important overall detoxification mechanism for the urinary bladder.

L49 ANSWER 51 OF 103 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN

L49 ANSWER 53 OF 103 MEDLINE on STN DUPLICATE 33
AB Human hydroxysteroid **sulfotransferase**, human **phenol** -sulfating form of **phenol sulfotransferase**, rat hydroxysteroid **sulfotransferase** a and rat **phenol sulfotransferase** IV were expressed in Escherichia coli. Cytosol preparations of transformed bacteria were used as activating systems in **mutagenicity** tests with Salmonella typhimurium TA98. All test compounds, 1-hydroxymethylpyrene, 2-hydroxymethylpyrene, 1-(1-pyrenyl)ethanol, 9-hydroxymethylanthracene, 7-hydroxymethyl-12-methylbenz[a]anthracene and 4H-cyclopenta[def]chrysen-4-ol, were activated by both hydroxysteroid **sulfotransferases** investigated. However, 1-(1-pyrenyl)ethanol was 67-fold more efficiently activated by the human enzyme, whereas 7-hydroxymethyl-12-methylbenz[a]anthracene was 27-fold more efficiently activated by the rat enzyme. The **phenol sulfotransferases** showed relatively low activities with the benzylic alcohols investigated. The only exception was 4H-cyclopenta[def]chrysen-4-ol, which was activated efficiently by rat **phenol sulfotransferase** IV. We had previously tested the ability of rat and human hepatic cytosol preparations to activate the same compounds. The results of a statistical analysis suggest that the activities of human hydroxysteroid **sulfotransferase**, rat hydroxysteroid **sulfotransferase** a and **phenol sulfotransferase** IV can account for a substantial portion of the activation of benzylic alcohols in human, female rat and male rat liver, respectively.

L49 ANSWER 63 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
AB Sulphation of the genotoxic compounds N-hydroxy-4-aminobiphenyl (N-OH-4ABP) and N-hydroxy-4-acetylaminobiphenyl (N-OH-4AABP) was determined in cytosolic preparations of human foetal, neonatal and adult liver and foetal and neonatal adrenal gland. Sulphotransferase (ST) activity capable of sulphating these compounds was present in foetal liver and adrenal gland by 14 weeks of gestation. Sulphation of N-OH-4ABP was higher in foetal and neonatal adrenal cytosol than was sulphation of N-OH-4AABP and in general, N-OH-4ABP ST activity was also greater than that towards 1-naphthol. In foetal and neonatal liver cytosol the sulphation of N-OH-4ABP was also higher than that of N-OH-4AABP (approximately 2-fold). In adult liver cytosols, however, N-OH-4AABP ST activity was higher than that for N-OH-4ABP and 1-naphthol sulphation. Aromatic hydroxylamines and hydroxamic acids are known to be converted by sulphotransferase into reactive, electrophilic compounds capable of reacting with DNA. Our data show that the human foetus and neonate have the capacity to sulphate these compounds and thus is able to produce the reactive **mutagenic** metabolites. Therefore, this class of genotoxic compounds may be bioactivated by humans during development-a time when they are most vulnerable to the effects of genotoxins.

L49 ANSWER 74 OF 103 MEDLINE on STN DUPLICATE 46
AB Cyclopenta[c,d]pyrene, a ubiquitous environmental and occupational pollutant, has been reported to be metabolically activated through

epoxidation at the 3,4 double bond in the cyclopenta ring to produce an electrophilic and **mutagenic** cyclopenta[c,d]pyrene-3,4-epoxide. 4-Hydroxy-3,4-dihydrocyclopenta[c,d]-pyrene (4-HDCPP) and 3,4-dihydroxy-3,4-dihydrocyclopenta[c,d]pyrene (3,4-DHDCPP) are known to be major metabolites of cyclopenta[c,d]pyrene, which appear to be derived from cyclopenta[c,d]pyrene-3,4-epoxide. The present study was undertaken to determine whether 4-HDCPP or 3,4-DHDCPP can be further activated via the formation of reactive benzylic sulfuric acid ester metabolites. Thus, when 4-HDCPP or 3,4-DHDCPP was incubated with calf thymus DNA in the presence of rodent liver cytosol and the sulfo group donor, 3'-phosphoadenosine-5'-phosphosulfate, a significant covalent DNA binding was observed. This cytosol- and 3'-phosphadenosine-5'-phosphosulfate-dependent DNA binding was inhibited by 2,6-dichloro-4-nitrophenol and dehydroepiandrosterone, suggesting the involvement of both **phenol** and hydroxysteroid **sulfotransferases** in the activation of 4-HDCPP and 3,4-DHDCPP. A gender difference was observed for the hepatic cytosolic **sulfotransferase** activity for 4-HDCPP in rats (i.e., male > female). Of the two isomers of 3,4-DHDCPP, the trans-diol produced DNA adducts to a much greater extent than did the cis counterpart by **sulfotransferase.** 4-HDCPP and 3,4-DHDCPP were also **mutagenic** toward bacteria in the presence of hepatic cytosol and 3'-phosphadenosine-5'-phosphosulfate. The chemically synthesized sulfuric acid ester 4-sulfooxy-3,4-DCPP was directly **mutagenic** without any activation system. The data from this study suggest that **sulfotransferase** plays an important role in the activation of those secondary benzylic hydroxyl metabolites derived from cyclopenta[c,d]pyrene-3,4-epoxide and, possibly, from epoxides of other aromatic hydrocarbons.

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L49 ANSWER 83 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

AB Our experiments were performed to determine whether human liver, like that of other mammals, could catalyze the N-sulfation of an arylamine, 2-naphthylamine (2-NA) and, if so, whether this reaction might be catalyzed by one or both of the two known forms of human **phenol sulfotransferase** (**PST**). One form of **PST** is thermostable (TS) and catalyzes the sulfation of "simple' **phenols** such as p-nitrophenol, while the other form is thermolabile (TL) and catalyzes the sulfate conjugation of **phenolic** monoamines such as dopamine. When 2-NA that was not contaminated with 2-naphthol was used as substrate, human hepatic cytosol could catalyze the N-sulfation of 2-NA with an apparent K(m) of 322-mu-M. However, substrate kinetics of the sulfate donor for the reaction, 3'-phosphoadenosine-5'-phosphosulfate, were biphasic, with estimated apparent K(m) values of 0.13 and 2.2-mu-M for high and low affinity activities, respectively. Human liver arylamine N-**sulfotransferase** (AANST) activity was similar to that of TS but not TL **PST** with regard to thermal stability, inhibition by 2,6-dichloro-4-nitrophenol (DCNP), and regulation among individuals. For example, average temperatures that produced 50% inactivation of TL **PST**, TS **PST**, and AANST activities, measured with both 0.05 and 1.0 mM 2-NA as substrate, were 35.0, 40.5, 40.3 and 40.5-degrees-C, respectively. IC50 values for the inhibition by DCNP of TL **PST**, TS **PST**, and AANST, measured with 0.05 and 1.0 mM 2-NA as substrate, were 110, 1.8, 1.3, and 4.0-mu-M, respectively. In addition, in cytosolic preparations from 20 individual human liver samples, there was a highly significant correlation between TS **PST** activity and AANST activity measured with both 0.05 mM 2-NA and 1.0 mM 2-NA as substrate ($r(s) = 0.899$, $p < 0.0001$ and $r(s) = 0.934$, $p < 0.0001$, respectively). However, there was not a significant correlation between TL **PST** activity and AANST measured with 0.05 mM and 1.0 mM 2-NA as substrate ($r(s) = 0.257$, $p = 0.278$ and $r(s) = 0.268$, $p = 0.258$, respectively). Finally, partially purified TS **PST**, but not

partially purified TL **PST**, catalyzed the N-sulfation of 2-NA. However, substrate kinetic studies once again indicated that the partially purified TS **PST** preparation might contain two AANST activities. In summary, our results were compatible with the conclusion that TS **PST** was the major enzyme responsible for catalyzing the N-sulfation of 2-NA in human liver cytosol. However, **sulfotransferase**(s) other than TS **PST** might also play a role in this reaction.

L49 ANSWER 89 OF 103 MEDLINE on STN DUPLICATE 51
AB The **phenol**-sulfating form of **phenol sulfotransferase** (P-**PST**) was purified and characterized from human liver cytosol using DEAE-cellulose, Sephacryl S-200, and 3',5'-diphosphoadenosine-agarose affinity chromatography. During the purification procedure, P-**PST** was resolved from the monoamine-sulfating form of **phenol sulfotransferase** (M-**PST**) and dehydroepiandrosterone **sulfotransferase**, which are also present in human liver cytosol. P-**PST** activity was purified 560-fold as compared to liver cytosol and the purified enzyme possessed a specific activity of 340 nmol **phenol** sulfated per minute per milligram protein. Enzymatically active P-**PST** has an apparent molecular size of 68,000 Da as determined by Sephacryl S-200 chromatography and a subunit molecular weight of 32,000 Da as determined by sodium dodecyl sulfate-polyacrylamide gel electrophoresis, suggesting that P-**PST** exists in vivo as a homodimer. Antibodies raised to human platelet M-**PST** cross-reacted strongly with pure P-**PST** suggesting the two PSTs are structurally closely related. Two types of P-**PST** activity have been identified in different human livers by their thermostability and elution during anion-exchange chromatography. Each of the livers examined possessed only one type of P-**PST** activity. Both types of P-**PST** were shown to possess the same subunit molecular weight and immunoreactivity, whereas the differences in thermostability of the two P-**PST** activities appeared to be related to the method of preparation of liver cytosol. Both types of P-**PST** activity were inhibited to similar extents by incubation with 50 microM N-ethylmaleimide or 5 mM phenylglyoxal. These results suggest that the two types of P-**PST** in different human livers are very similar and probably represent different **allelic** forms of the enzyme.

L49 ANSWER 100 OF 103 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 56

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          4391 ALLOZYM?
         63310 POLYMORPH?
L64         47 (L24 OR L52) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

FILE 'BIOTECHDS'
           160 SULFOTRANSFERASE#
         12476 PHENOL?
           181 PST
         45885 MUTA?
          8143 ALLEL?
         15686 VARIANT#
            36 ALLOZYM?
          9321 POLYMORPH?
L65         25 (L24 OR L53) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)
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FILE 'BIOSIS'
          3382 SULFOTRANSFERASE#
         73196 PHENOL?
          2858 PST
        557139 MUTA?
        127111 ALLEL?
        114609 VARIANT#
          8971 ALLOZYM?
        193949 POLYMORPH?
L66        194 (L24 OR L54) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

FILE 'EMBASE'
          2715 SULFOTRANSFERASE#
         35457 PHENOL?
          1599 PST
        430474 MUTA?
         90219 ALLEL?
         98356 VARIANT#
          1080 ALLOZYM?
        135069 POLYMORPH?
L67        158 (L24 OR L55) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

FILE 'HCAPLUS'
          3820 SULFOTRANSFERASE#
        411274 PHENOL?
          2887 PST
        524593 MUTA?
        103442 ALLEL?
        111646 VARIANT#
          2948 ALLOZYM?
        185887 POLYMORPH?
L68        222 (L24 OR L56) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

FILE 'NTIS'
            18 SULFOTRANSFERASE#
          6655 PHENOL?
           132 PST
         10096 MUTA?
           612 ALLEL?
          4656 VARIANT#
            29 ALLOZYM?
          1634 POLYMORPH?
L69           5 (L24 OR L57) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

FILE 'ESBIOBASE'
          1176 SULFOTRANSFERASE#
         31341 PHENOL?
           632 PST
        263942 MUTA?
         59141 ALLEL?
         47320 VARIANT#
          3673 ALLOZYM?
         70396 POLYMORPH?
L70        136 (L24 OR L58) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

FILE 'BIOTECHNO'
          1049 SULFOTRANSFERASE#
         10046 PHENOL?
           687 PST
        242571 MUTA?
```

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         55235 ALLEL?
         41198 VARIANT#
          1661 ALLOZYM?
         71286 POLYMORPH?
L71         83 (L24 OR L59) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

FILE 'WPIDS'
           143 SULFOTRANSFERASE#
        125746 PHENOL?
           502 PST
         29252 MUTA?
          7412 ALLEL?
         27746 VARIANT#
             7 ALLOZYM?
          8484 POLYMORPH?
L72          6 (L24 OR L60) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

TOTAL FOR ALL FILES
L73       1268 (L24 OR L61) AND (MUTA? OR ALLEL? OR VARIANT# OR ALLOZYM? OR
               POLYMORPH?)

=> s l73 not 2004-2006/py
FILE 'MEDLINE'
       1430981 2004-2006/PY
                 (20040000-20069999/PY)
L74        117 L62 NOT 2004-2006/PY

FILE 'SCISEARCH'
       2627808 2004-2006/PY
                 (20040000-20069999/PY)
L75        144 L63 NOT 2004-2006/PY

FILE 'LIFESCI'
        210840 2004-2006/PY
L76         33 L64 NOT 2004-2006/PY

FILE 'BIOTECHDS'
         61513 2004-2006/PY
L77         12 L65 NOT 2004-2006/PY

FILE 'BIOSIS'
       1097231 2004-2006/PY
L78        134 L66 NOT 2004-2006/PY

FILE 'EMBASE'
       1216904 2004-2006/PY
L79        108 L67 NOT 2004-2006/PY

FILE 'HCAPLUS'
       2740644 2004-2006/PY
L80        134 L68 NOT 2004-2006/PY

FILE 'NTIS'
         28990 2004-2006/PY
L81          4 L69 NOT 2004-2006/PY

FILE 'ESBIOBASE'
        715825 2004-2006/PY
L82         90 L70 NOT 2004-2006/PY

FILE 'BIOTECHNO'
           586 2004-2006/PY
L83         82 L71 NOT 2004-2006/PY
```

```
FILE 'WPIDS'
       2652763 2004-2006/PY
L84          1 L72 NOT 2004-2006/PY

TOTAL FOR ALL FILES
L85        859 L73 NOT 2004-2006/PY

=> s l85 and 1999-2003/py
FILE 'MEDLINE'
       2584873 1999-2003/PY
                 (19990000-20039999/PY)
L86         67 L74 AND 1999-2003/PY

FILE 'SCISEARCH'
       5052295 1999-2003/PY
                 (19990000-20039999/PY)
L87         85 L75 AND 1999-2003/PY

FILE 'LIFESCI'
        572693 1999-2003/PY
L88         17 L76 AND 1999-2003/PY

FILE 'BIOTECHDS'
         97694 1999-2003/PY
L89         11 L77 AND 1999-2003/PY

FILE 'BIOSIS'
       2847812 1999-2003/PY
L90         82 L78 AND 1999-2003/PY

FILE 'EMBASE'
       2316144 1999-2003/PY
L91         61 L79 AND 1999-2003/PY

FILE 'HCAPLUS'
       5003186 1999-2003/PY
L92         80 L80 AND 1999-2003/PY

FILE 'NTIS'
        103448 1999-2003/PY
L93          4 L81 AND 1999-2003/PY

FILE 'ESBIOBASE'
       1453117 1999-2003/PY
L94         56 L82 AND 1999-2003/PY

FILE 'BIOTECHNO'
        610762 1999-2003/PY
L95         43 L83 AND 1999-2003/PY

FILE 'WPIDS'
       4344996 1999-2003/PY
L96          1 L84 AND 1999-2003/PY

TOTAL FOR ALL FILES
L97        507 L85 AND 1999-2003/PY

=> dup rem l97
PROCESSING COMPLETED FOR L97
L98         152 DUP REM L97 (355 DUPLICATES REMOVED)

=> d tot

L98  ANSWER 1 OF 152  HCAPLUS  COPYRIGHT 2006 ACS on STN
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Avail.: Metadata on Internet Documents, Order No. 19233
From: Metadata Internet Doc. [Ger. Diss.] 2003, (D1018-4), No pp. given
AU Pabel, Ulrike
AN 2005:1147620 HCAPLUS
DN 144:344893

L98 ANSWER 2 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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From: Diss. Abstr. Int., B 2004, 64(12), 5937
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L98 ANSWER 3 OF 152 NTIS COPYRIGHT 2006 NTIS on STN
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L98 ANSWER 5 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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SO Jpn. Kokai Tokkyo Koho, 14 pp.
CODEN: JKXXAF
IN Suiko, Masahito
AN 2003:761902 HCAPLUS
DN 139:273220

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2003274997	A2	20030930	JP 2002-79119	20020320 <--

L98 ANSWER 6 OF 152 MEDLINE on STN DUPLICATE 1
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L98 ANSWER 8 OF 152 MEDLINE on STN DUPLICATE 3
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L98 ANSWER 11 OF 152 MEDLINE on STN DUPLICATE 5
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L98 ANSWER 12 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 17 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 20 OF 152 MEDLINE on STN DUPLICATE 9
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L98 ANSWER 21 OF 152 MEDLINE on STN DUPLICATE 10
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L98 ANSWER 24 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 25 OF 152 MEDLINE on STN DUPLICATE 12
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L98 ANSWER 28 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 29 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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L98 ANSWER 30 OF 152 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 15
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L98 ANSWER 33 OF 152 MEDLINE on STN DUPLICATE 16
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L98 ANSWER 35 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 36 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 38 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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SO International Journal of Cancer (**2003**), Volume Date 2004, 108(1), 97-103
CODEN: IJCNAW; ISSN: 0020-7136
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

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L98 ANSWER 83 OF 152 MEDLINE on STN DUPLICATE 39
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L98 ANSWER 84 OF 152 MEDLINE on STN DUPLICATE 40
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L98 ANSWER 85 OF 152 MEDLINE on STN DUPLICATE 41
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L98 ANSWER 86 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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CODEN: 69EDZN; ISBN: 0-7484-0864-9
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DN 140:196577

L98 ANSWER 87 OF 152 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
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L98 ANSWER 88 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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Meeting Info.: 92nd Annual Meeting of the American Association for Cancer Research. New Orleans, LA, USA. March 24-28, 2001.
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L98 ANSWER 89 OF 152 MEDLINE on STN DUPLICATE 43
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L98 ANSWER 90 OF 152 MEDLINE on STN DUPLICATE 44
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Journal code: 8008055. ISSN: 0143-3334.
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L98 ANSWER 91 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 93 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 94 OF 152 MEDLINE on STN DUPLICATE 46
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Journal code: 9211735. ISSN: 0960-314X.
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L98 ANSWER 95 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 96 OF 152 MEDLINE on STN DUPLICATE 47
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Ref: 98
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L98 ANSWER 98 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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AN 2001:84855 SCISEARCH

L98 ANSWER 99 OF 152 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
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AN 2000-08749 BIOTECHDS
PI WO 2000020605 **13 Apr 2000**

L98 ANSWER 100 OF 152 MEDLINE on STN DUPLICATE 49
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Journal code: 0370623. ISSN: 0006-2960.
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AN 2001116745 MEDLINE

L98 ANSWER 101 OF 152 MEDLINE on STN DUPLICATE 50
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L98 ANSWER 102 OF 152 MEDLINE on STN DUPLICATE 51
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L98 ANSWER 103 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 52
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L98 ANSWER 104 OF 152 MEDLINE on STN DUPLICATE 53
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L98 ANSWER 105 OF 152 MEDLINE on STN DUPLICATE 54
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Journal code: 8008055. ISSN: 0143-3334.
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L98 ANSWER 106 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 107 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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ISSN: 0892-6638.
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AN 2000:554158 SCISEARCH

L98 ANSWER 108 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 109 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 110 OF 152 MEDLINE on STN DUPLICATE 55
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SO Carcinogenesis, (**2000 Jul**) Vol. 21, No. 7, pp. 1347-54.
Journal code: 8008055. ISSN: 0143-3334.
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L98 ANSWER 111 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 112 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

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AN 2000:266005 BIOSIS

L98 ANSWER 113 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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ISSN: 0197-016X.
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L98 ANSWER 114 OF 152 MEDLINE on STN DUPLICATE 56
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SO Pharmacogenetics, (**2000 Dec**) Vol. 10, No. 9, pp. 789-97.
Journal code: 9211735. ISSN: 0960-314X.
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AN 2001201563 MEDLINE

L98 ANSWER 115 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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CODEN: ZLYZE9; ISSN: 1001-6821
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AN 2001:332013 HCAPLUS
DN 135:266437

L98 ANSWER 116 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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AN 2000:488709 BIOSIS

L98 ANSWER 117 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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CODEN: MUREAV; ISSN: 0027-5107
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AN 2000:246355 HCAPLUS
DN 133:87505

L98 ANSWER 118 OF 152 MEDLINE on STN DUPLICATE 57
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SO Carcinogenesis, **(2000 Feb)** Vol. 21, No. 2, pp. 295-9.
Journal code: 8008055. ISSN: 0143-3334.
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L98 ANSWER 119 OF 152 MEDLINE on STN DUPLICATE 58
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Journal code: 0372516. ISSN: 0006-291X.
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L98 ANSWER 120 OF 152 MEDLINE on STN DUPLICATE 59
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Journal code: 0417615. ISSN: 0340-5761.
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AN 2001103760 MEDLINE

L98 ANSWER 121 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
TI Genetic **polymorphism** of human tissue **phenol sulfotransferases**
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CODEN: YADOEL; ISSN: 0916-1139
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AN 2000:590725 HCAPLUS
DN 133:332517

L98 ANSWER 122 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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AN 2000:186992 SCISEARCH

L98 ANSWER 123 OF 152 MEDLINE on STN DUPLICATE 60
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SO Pharmacogenetics, **(2000 Mar)** Vol. 10, No. 2, pp. 163-9.
Journal code: 9211735. ISSN: 0960-314X.
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AN 2000222641 MEDLINE

L98 ANSWER 124 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 61
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SO ANNUAL REVIEW OF PHARMACOLOGY AND TOXICOLOGY, **(2000)** Vol. 40, pp. 159-176.
ISSN: 0362-1642.
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L98 ANSWER 125 OF 152 MEDLINE on STN DUPLICATE 62
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Journal code: 0227276. ISSN: 0009-2797.
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AN 2001120316 MEDLINE

L98 ANSWER 126 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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ISSN: 0065-7727.
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L98 ANSWER 127 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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SO Drug Metabolism Reviews, (**2000**) Vol. 32, No. Supplement 1, pp. 97. print.
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L98 ANSWER 128 OF 152 MEDLINE on STN
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Journal code: 0372430. ISSN: 0003-9861.
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L98 ANSWER 129 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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AN 2000:419181 BIOSIS

L98 ANSWER 130 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 131 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
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CODEN: 69CLAC
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AN 2000:327095 HCAPLUS

L98 ANSWER 132 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 133 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
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CODEN: ACSRAL. ISSN: 0065-7727.
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AN 2000:190520 BIOSIS

L98 ANSWER 134 OF 152 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
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recombinant **phenol-sulfotransferase** production **mutation** detection via vector-mediated gene transfer and expression in host cell for disease diagnosis and in animal model
AU Guida M; Kurth J
AN 2000-04354 BIOTECHDS
PI WO 9964630 **16 Dec 1999**

L98 ANSWER 135 OF 152 MEDLINE on STN DUPLICATE 64
TI X-ray crystal structure of human dopamine **sulfotransferase, SULT1A3.** Molecular modeling and quantitative structure-activity relationship analysis demonstrate a molecular basis for **sulfotransferase** substrate specificity.
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L98 ANSWER 136 OF 152 MEDLINE on STN DUPLICATE 65
TI Analysis of the substrate specificity of human **sulfotransferases** **SULT1A1** and **SULT1A3**: site-directed **mutagenesis** and kinetic studies.
SO Biochemistry, **(1999 Aug 10)** Vol. 38, No. 32, pp. 10474-9.
Journal code: 0370623. ISSN: 0006-2960.
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L98 ANSWER 137 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 138 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
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L98 ANSWER 152 OF 152 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 72
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ANSWER SET L98 HAS BEEN SAVED AS 'SULT1A1624/A'

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33,37-40,44,47,53-55,60,64-66,68,70-78,80,83,86-90,92,94-96,99,102,104,105,108,111,113,114,120-125,132,134,139,140,141,144,146,149

L73 ANSWER 33 OF 152 MEDLINE on STN DUPLICATE 16
AB Gene-environment interactions are hypothesized to be major contributors to susceptibility to environmental carcinogens and interindividual variability in cancer risk. We present findings on associations between genetic susceptibility due to inherited **polymorphisms** of the Phase II detoxification enzyme sulfotransferase 1A1 (**SULT1A1**), breast cancer risk, and polycyclic aromatic hydrocarbon (PAH)-DNA adducts. A hospital based case-control study was conducted at the New York-Presbyterian Medical Center (NYPMC). The study utilized two control groups: one comprised of women with benign breast disease (BBD) and the other comprised of women visiting NYPMC for routine gynecologic checkups (healthy controls). Blood samples were collected from cases and controls; and breast tissue from pathology blocks was collected from cases (tumor and non-tumor tissue) and BBD controls (benign tissue). PAH-DNA adduct levels were measured by immunohistochemistry in breast tissue samples, and the **SULT1A1** (Arg/His) **polymorphism** at codon 213 was determined by PCR RFLP analyses using DNA from white blood cells. Increasing number of His **alleles** was modestly associated with breast cancer case-control status, when cases were compared to healthy controls (p for trend = 0.08), when cases were compared to BBD controls (p

for trend = 0.08) and when cases were compared to both control groups combined (p for trend = 0.07). Contrary to our hypothesis PAH-DNA adduct levels in breast tissue were not associated with **SULT1A1** genotype. Our findings are consistent with a prior report that the Arg/His **polymorphism** in **SULT1A1** is associated with breast cancer risk.

L73 ANSWER 37 OF 152 MEDLINE on STN DUPLICATE 17
AB Sulfotransferase (SULT) 1A1 detoxifies and bioactivates a broad spectrum of substrates including xenobiotics. It has been suggested that the **SULT1A1** his (histidine) **allele**, which is caused by a his for arg (arginine) substitution due to a G-->A transition at codon 213, carries a significantly higher risk for women to develop breast cancer. We investigated the association between the **SULT1A1** arg/his genotype and esophageal cancer in men, 187 cases of esophageal squamous cell carcinoma and 308 controls from 3 medical centers in Taiwan. Cigarette smoking, areca chewing and alcohol consumption were the major risks for developing esophageal cancer. The frequencies of arg/his in cases and controls were 27.8% (52/187) and 11.0% (34/308), respectively (p < 0.0001). No subjects carried his/his. After adjusting for substance use and other covariates, individuals with arg/his had a 3.53-fold higher risk (95% CI = 2.12-5.87) of developing esophageal cancer than those with arg/arg. Unexpectedly, this positive association was found to be even stronger (adjusted OR = 4.04-4.80) among non-smokers, non-drinkers or non-chewers. Our findings suggest that the **SULT1A1** his(213) **allele** is important in the development of esophageal cancer in men.

L73 ANSWER 38 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB Cigarette smoke contains polycyclic hydrocarbons and arylamines that may both be activated by **sulfotransferase**, encoded by **SULT1A1.** A genetic **polymorphism** leads to an Arg213His substitution, thereby decreasing enzyme activity and stability and might thus modify the association between smoking and colorectal adenomas. We investigated this in a Dutch case-control study. Addnl., the authors evaluated potential roles of epoxide hydrolase (EPHX), N-acetyltransferases (NAT1 and NAT2) and glutathione S-transferases (GSTM1 and GSTT1). The data anal. included 431 adenoma cases and 432 polyp-free controls (54% women; mean age, 54.6 yr) enrolled at endoscopy in 8 Dutch hospitals between 1997 and 2000. All participants provided data on smoking habits and blood for DNA isolation. Genotyping was performed using appropriate PCR -restriction fragment length **polymorphism** procedures. Multivariate models included age, sex, endoscopy indication, consumption of snacks and alc. and, if appropriate, daily smoking dose or smoking duration. Smoking increased colorectal adenoma risk, most importantly by duration. Smoking for more than 25 yr more than doubled adenoma risk (OR = 2.4, 95% CI = 1.4-4.1) compared to never smoking. Combinations of **SULT1A1** fast sulfation (*1/*1) and of NAT2 slow acetylation with smoking resulted in a 4 times higher risk of adenomas compared to never smokers with other inherited gene **variants**, although there was no statistically significant effect modification. We found no clear effects of the other genetic **polymorphisms** on the association between smoking and adenomas. We conclude that smoking increases risk of colorectal adenomas and that **SULT1A1** and NAT2 only modestly modify this association

L73 ANSWER 39 OF 152 MEDLINE on STN DUPLICATE 18
AB Sulfotransferase (SULT) enzymes play an important role in the detoxification, metabolism and bioactivation of numerous xenobiotics, many dietary and environmental **mutagens**, drugs, neurotransmitters and hormones. The genes for **SULT1A1** and **SULT1A2** contain common genetic **polymorphisms** that are associated with individual variations in the level of enzyme activities as well as variations of biochemical and physical properties. We developed a PCR-RFLP method to

analyze the frequencies of **SULT1A1** and **SULT1A2** **alleles** among cancerous patients and normal controls in Taiwan. The results showed that **SULT1A1***1 and **SULT1A2***1 were in positive linkage disequilibrium. Neither **SULT1A1***3 nor **SULT1A2***3 were found in this study. The frequencies of **SULT1A1***2 and **SULT1A2***2 for hepatic, colon, lung, oral, gastric, renal and cervical cancerous patients were 3.95, 5.56, 4.92, 3.84, 2.70, 7.41 and 4.50%, respectively. No statistical significance was found for these cancer patients after comparison with normal controls (4.0%) for the **allelic** frequencies of **SULT1A1***2 and **SULT1A2***2.

L73 ANSWER 40 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

L73 ANSWER 44 OF 152 NTIS COPYRIGHT 2006 NTIS on STN
AB The purpose of these studies is to elucidate the pharmacogenetic factors that contribute to variation in human response to tamoxifen (TAM) and raloxifene (RAL). We had previously identified and partially characterized common genetic **polymorphisms** in two human drug-metabolizing genes, **SULT1A1** and UGT1A6. We hypothesized that these **polymorphisms** contributed to variation in TAM or RAL metabolism. These studies were divided into three aims with the purpose of: (1) biochemically characterizing the contribution of these enzymes to the metabolism of TAM and RAL; (2) developing cell model systems to study **allele**- specific differences in cellular response to these molecules and; (3) perform a clinical pharmacogenetic study to evaluate the association of common genetic **polymorphisms** in drug metabolizing genes with variable clinical response to TAM. Thus far we have determined that **SULT1A1** and UGT1A6 contribute to the inactivation of 4-hydroxytamoxifen (OHT), the active metabolite of TAM, and that a separate enzyme, UGT1A9 catalyzed the glucuronidation of RAL. We have determined genotype/phenotype correlation for UGT1A6 **alleles** in a bank of human liver tissue and have generated HEK 293 cell lines that stably express each of the four UGT1A6 **allozymes.** The UGT1A6 (star)2 **allozyme**, when expressed homozygously, is associated with high UGT1A6 activity. We established MCF-7 breast cancer cell lines stably expressing the wild type and **variant SULT1A1 alleles** and have measured **allele**-specific differences in the response of these cells to estrogens and OHT. These studies suggest that pharmacogenetic factors might contribute to variable cellular response to antiestrogens.

L73 ANSWER 47 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB The present invention relates to genetic polymorphism data, compns. and methods for detecting genetic polymorphisms, methods for evaluating drugs using genetic polymorphisms, and screening methods for drugs. Thus, 7669 sep. single nucleotide polymorphisms (SNP) are provided in human genes encoding drug-metabolizing enzymes. In some embodiments, a drug-metabolizing enzyme is at least one of the following: epoxide hydrolase, methyltransferase, N-acetyltransferase, sulfotransferase, quinone oxidoreductase, glutathione S-transferase, UDP-glycosyltransferase, aldehyde dehydrogenase, alc. dehydrogenase, esterase, NDUF, cytochrome P 450, and ATP-binding cassette. In one example, a correlation is demonstrated between optimal amts. of azathioprine (an immunosuppressive agent) and various combinations of the alleles at the 868th SNP of intron 3 of thiopurine S-methyltransferase gene (G or T alleles) and the 2682nd SNP of intron 3 (C or A alleles). [This abstract record is one of two records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L73 ANSWER 53 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB A review. Common genetic variations may, in large part, explain genetic predispositions to common diseases and individual differences in

pharmacol. responsiveness. Single-nucleotide **polymorphisms** (SNPs) are the most frequent type of genetic variation, and are thought to be present every several hundred bases, on average, throughout the human genome. SNPs can provide medically important information through (1) their contribution to high-d. maps for studies of susceptibility to common diseases; (2) provision of genetic data for personalized medical service; and (3) identification of genes associated with the efficacy and side-effect of drugs. We have been focusing on genomic loci that encode various enzymes and transporters involved in the metabolism of drugs, and have described more than 5,500 SNP and other variations. Our collection of human variations should prove useful for investigations designed to detect assocns. between genetic variations and common diseases or responsiveness to drug therapy. In this review, we introduce the recent progress and future direction of human genome anal. and its impact on medicine.

L73 ANSWER 54 OF 152 MEDLINE on STN DUPLICATE 25

AB BACKGROUND: Human **sulfotransferase** 1A1 (**SULT1A1**) catalyzes the sulfation of a variety of **phenolic** and estrogenic compounds, including 4-hydroxytamoxifen (4-OH TAM), the active metabolite of tamoxifen. A functional **polymorphism** in exon 7 of the **SULT1A1** gene (**SULT1A1***2) has been described that generates an enzyme that has approximately twofold lower activity and is less thermostable than that of the common **allele SULT1A1** *1. We investigated the hypothesis that that high sulfation activity would increase the elimination of 4-OH TAM by examining whether the presence of this **polymorphism** affects the efficacy of tamoxifen therapy. METHODS: We examined the relationship between the **SULT1A1***2 **allele** and survival in a cohort of 337 women with breast cancer who received tamoxifen (n = 160) or who did not (n = 177). **SULT1A1** genotype was determined by restriction fragment **polymorphism** analysis. Patient survival was evaluated according to **SULT1A1** genotype using Kaplan-Meier survival functions. Hazard ratios (HRs) were calculated from adjusted Cox proportional hazards modeling. All statistical tests were two-sided. RESULTS: Among tamoxifen-treated patients, those who were homozygous for the **SULT1A1***2 low-activity **allele** had approximately three times the risk of death (HR = 2.9, 95% confidence interval [CI] = 1.1 to 7.6) as those who were homozygous for the common **allele** or those who were heterozygous (**SULT1A1***1/*2). Among patients who did not receive tamoxifen, there was no association between survival and **SULT1A1** genotype (HR = 0.7, 95% CI = 0.3 to 1.5). CONCLUSIONS: Sulfation of 4-OH TAM provides a previously unanticipated benefit, possibly due to alterations in the bioavailability of the active metabolite or to undefined estrogen receptor-mediated events. These data alternatively suggest that variability in the metabolism of tamoxifen may affect its efficacy.

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L73 ANSWER 60 OF 152 MEDLINE on STN DUPLICATE 27

AB 1. Sulphotransferases are a superfamily of enzymes involved in both detoxification and bioactivation of endogenous and exogenous compounds. The arylsulphotransferase **SULT1A1** has been implicated in a decreased activity and thermostability when the wild-type arginine at position 213 of the coding sequence is substituted by a histidine. **SULT1A1** is the isoform primarily associated with the conversion of dietary N-OH arylamines to DNA binding adducts and is therefore of interest to determine whether this **polymorphism** is linked to colorectal cancer. 2. Genotyping, using polymerase chain reaction-restriction fragment length **polymorphism** (PCR-RFLP) analysis, was performed using DNA samples of healthy control subjects (n = 402) and patients with histologically proven colorectal cancer (n = 383). Both control and test populations possessed similar frequencies for the **mutant allele** (32.1 and 31%, respectively; P = 0.935).

Results were not altered when age and gender were considered as potential confounders in a logistic regression analysis. 3. Examination of the sulphonating ability of the two **allozymes** with respect to the substrates p-nitrophenol and paracetamol showed that the affinity and rate of sulphonation was unaffected by substitution of arginine to histidine at position 213 of the amino acid sequence. 4. From this study, we conclude that the **SULT1A1** R213H **polymorphism** is not linked with colorectal cancer in this elderly Australian population.

L73 ANSWER 64 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

L73 ANSWER 65 OF 152 MEDLINE on STN DUPLICATE 30

L73 ANSWER 66 OF 152 MEDLINE on STN DUPLICATE 31

AB Carcinogenic aromatic amines such as 4-aminobiphenyl, which is contained in tobacco smoke, are one of the causal factors of urothelial epithelial cancers. 4-Aminobiphenyl has been shown to be bioactivated through N-hydroxylation by hepatic cytochrome (CYP) 1A2 and subsequently through O-sulfation and O-acetylation by **phenol** sulfating **sulfotransferase**, ST1A3 (**SULT1A1**), and arylamine N-acetyltransferase, NAT2, respectively. In a case-control study for urothelial epithelial cancers, low activity **alleles** of NAT2 are overall high-risk **alleles** (OR 2.11; 95% CI 1.08-4.26). Wild-type ST1A3*1 ((213)Arg) **alleles** were slightly overrepresented in nonsmoking urothelial cancer patients (82.6% vs. 69.7%) and in smoking cancer patients (76.7% and 74.3%) compared to a **variant** ST1A3*2 ((213)His) **allele.** In combination of ST1A3 and NAT2 genotypes for analyses of urothelial cancer risk, the highest OR of 2.45 (95% CI 1.04-5.98) was obtained with ST1A3*1 and NAT2 slow genotype among the 4 combinations. Recombinant ST1A3*1 enzyme showed a tendency of catalyzing higher in vitro 3'-phosphoadenosine 5'-phosphosulfate-dependent DNA adduct formation than ST1A3*2 (2.84 +/- 0.49 and 2.22 +/- 0.11 adducts/10(8) nucleotides). Combined analyses of different **alleles** of carcinogenic aromatic amine-activating phase II enzymes were applied to urothelial cancer risk for the first time and showed the highest risk combination of ST1A3 and NAT2 **alleles**
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L73 ANSWER 68 OF 152 MEDLINE on STN DUPLICATE 33

AB Members of the cytosolic sulfotransferase (SULT) superfamily catalyse the sulfation of a multitude of xenobiotics, hormones and neurotransmitters. Humans have at least 10 functional SULT genes, and a number of recent advances reviewed here have furthered our understanding of SULT function. Analysis of expression patterns has shown that sulfotransferases are highly expressed in the fetus, and SULTs may in fact be a major detoxification enzyme system in the developing human. The X-ray crystal structures of three SULTs have been solved and combined with **mutagenesis** experiments and molecular modelling, they have provided the first clues as to the factors that govern the unique substrate specificities of some of these enzymes. In the future these and other studies will facilitate prediction of the fate of chemicals metabolised by sulfation. Variation in sulfation capacity may be important in determining an individual's response to xenobiotics, and there has been an explosion in information on sulfotransferase **polymorphisms** and their functional consequences, including the influence of **SULT1A1** genotype on susceptibility to colorectal and breast cancer. Finally, the first gene knockout experiments with SULTs have recently been described, with the generation of estrogen sulfotransferase deficient mice in which reproductive capacity is compromised. Our improved understanding of these enzymes will have significant benefits in such diverse areas as drug design and development, cancer susceptibility, reproduction and development.

L73 ANSWER 70 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

L73 ANSWER 71 OF 152 MEDLINE on STN DUPLICATE 35
AB A case-control study of colorectal cancer, consisting of 157 cases and 380 controls matched by sex, ethnicity, decade of age and county of residence was performed to explore the associations between environmental exposure, metabolic **polymorphisms** and cancer risk. Participants were required to provide a blood sample, undergo caffeine phenotyping and complete an in-person interview that evaluated meat consumption, cooking methods and degree of doneness. A color atlas of foods cooked to different degrees of doneness was used to estimate food preparation techniques and food models were used to estimate serving portion sizes. Data was analyzed using a reference database of heterocyclic amine (HCA) exposure based on the food preferences chosen from the atlas. Data regarding individual food items cooked to different levels of doneness, as well as summary variables of foods and of food groups cooked to different degrees of doneness were also evaluated in a univariate analysis for association with colorectal cancer case status. Three measures of metabolic variation, hGSTA1 genotype, **SULT1A1** genotype and the phenotype for CYP2A6 were also evaluated for possible association with colon cancer.While higher exposure to HCAs was strongly associated with colorectal cancer risk, increased consumption of five red meats cooked well done or very well done produced comparable odds ratios (OR) for colorectal cancer risk (OR=4.36, 95% CI 2.08-9.60) for the highest quartile of exposure. Similarly, individuals in the most rapid CYP2A6 phenotype quartile showed an odds ratio (OR = 4.18, 95% CI 2.03-8.90). The ORs for the low activity hGSTA1 and low activity **SULT1A1** **alleles** were 2.0, 95% CI 1.0-3.7 and 0.6, 95% CI 0.3-1.1, respectively. Individual measures of specific HCAs provided little improvement in risk assessment over the measure of meat consumption, suggesting that exposure to other environmental or dietary carcinogens such as nitrosamines or undefined HCAs may contribute to colorectal cancer risk.

L73 ANSWER 72 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

L73 ANSWER 73 OF 152 MEDLINE on STN DUPLICATE 36
AB **SULT1A1** enzyme is a member of the sulfotransferase family that alters biological activities of numerous carcinogenic and **mutagenic** compounds through sulfation. A genetic **polymorphism** in the coding region of **SULT1A1** gene has been associated with modulated enzyme activity. There is a G-->A nucleotide **polymorphism** in **SULT1A1** gene that codes for an Arg-->His substitution, which results in decreased activity and thermal stability of the **SULT1A1** enzyme. Utilizing a case-control study design, we hypothesized that the **variant** **allele** of the **SULT1A1** gene may be associated with lung cancer risk. The PCR-RFLP assay was used to successfully genotype the **SULT1A1***2 **allele** (**variant** A-**allele**) in 463 Caucasian lung cancer cases and 485 frequency matched Caucasian controls. There was an overall significant difference between cases and controls when adjusted by sex and smoking status (adjusted OR=1.41, 95% CI: 1.04-1.91). The adjusted OR was higher for females (OR=1.64, 95% CI: 1.06-2.56) than for males (OR=1.23, 95% CI: 0.80-1.88). Furthermore, the risk was significantly higher in current smokers (OR=1.74, 95% CI: 1.08-2.29) and heavy smokers (OR=1.45, 95% CI: 1.05-2.00). Our results support the hypothesis that a genetic **polymorphism** in the **SULT1A1** gene may be associated with increased lung cancer risk.

L73 ANSWER 74 OF 152 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

L73 ANSWER 75 OF 152 NTIS COPYRIGHT 2006 NTIS on STN

AB The purpose of these studies is to elucidate the pharmacogenetic factors that contribute to variation in human response to tamoxifen (TAM) and raloxifene (RAL). We had previously identified and partially characterized common genetic **polymorphisms** in two human drug-metabolizing genes, **SULT1A1** and UGT1A1. We hypothesized that these **polymorphisms** contributed to variation in TAM or RAL metabolism. These studies were divided into three aims with the purpose of: (1) biochemically characterizing the contribution of these enzymes to the metabolism of TAM and RAL; (2) developing cell model systems to study **allele**- specific differences in cellular response to these molecules and; (3) perform a clinical pharmacogenetic study to evaluate the association of common genetic **polymorphisms** in drug metabolizing genes with variable clinical response to TAM. Thus far we have determined that **SULT1A1** and UGT1A6 contributed to the inactivation of 4-hydroxytamoxifen (OHT), the active metabolite of TAM, and that a separate enzyme, UGT1A9 catalyzed the glucuronidation of RAL. We established MCF-7 breast cancer cell lines stably expressing the wildtype and **variant** **SULT1A1 alleles** and have measured **allele** -specific differences in the response of these cells to estrogens and OHT. These studies suggest that pharmacogenetic factors might contribute to variable cellular response to antiestrogens.

L73 ANSWER 76 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to determine the hypersensitivity of individuals to a given agent, such as drug or other chemical, in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes associated with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes associated with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes associated with hypersensitivity. The expression of the genes predetd. to be associated with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and apparatus useful for identifying hypersensitivity in a subject are also disclosed.

L73 ANSWER 77 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB The present invention relates to methods for identifying and/or classifying patients with inflammatory bowel diseases (IBD), particularly patients with Crohn's disease or ulcerative colitis. Gene expression profiling shows broad and fundamental differences in the pathogenic mechanism of ulcerative colitis and Crohn's disease. The subject method is based on the findings that certain genes are differentially expressed in intestinal tissue of IBD patients compared with related normal cells, such as normal colon cells. That change can be used to identify or classify IBD cells by the upregulation and/or downregulation of expression of particular genes, alterations in protein levels or modification, or changes at the genomic level (such as **mutation**, methylation, etc), e.g., an event which is implicated in the pathol. of inflammatory bowel diseases.

L73 ANSWER 78 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB Methods for identifying and utilizing variances in genes relating to efficacy and safety of medical therapy and other aspects of medical therapy are described, including methods for selecting an effective treatment. [This abstract record is one of several records for this document necessitated by the large number of index entries required to fully

index the document and publication system constraints.].

L73 ANSWER 80 OF 152 MEDLINE on STN DUPLICATE 37
AB Sulphation is an important detoxification pathway for numerous xenobiotics; however, it also plays an important role in the metabolism and bioactivation of many dietary and environmental **mutagens**, including heterocyclic amines implicated in the pathogenesis of colorectal and other cancers. A major sulphotransferase (SULT) enzyme in humans, **SULT1A1**, is **polymorphic** with the most common **variant allele**, **SULT1A1***2, occurring at a frequency of about 32% in the Caucasian population. This **allele** codes for an **allozyme** with low enzyme activity and stability compared to the wild-type (**SULT1A1***1) enzyme, and therefore **SULT1A1** genotype may influence susceptibility to **mutagenicity** following exposure to heterocyclic amines and other environmental toxins. Previously, a significant association of **SULT1A1***1 genotype with old age has been observed, suggesting a 'chemoprotective' role for the high-activity phenotype. Here we have compared the frequencies of the most common **SULT1A1** **alleles** in 226 colorectal cancer patients and 293 previously described control patients. We also assessed whether **SULT1A1** genotype was related to various clinical parameters in the patient group, including Duke's classification, differentiation, site, nodal involvement and survival. There was no significant difference in **allele** frequency between the control and cancer patient populations, nor was there a significant association with any of the clinical parameters studied. However, when the age-related difference in **allele** frequency was considered, a significantly reduced risk of colorectal cancer (odds ratio = 0.47; 95% confidence interval = 0.27-0.83; P = 0.009), was associated with homozygosity for **SULT1A1***1 in subjects under the age of 80 years. These results suggest that the high activity **SULT1A1***1 **allozyme** protects against dietary and/or environmental chemicals involved in the pathogenesis of colorectal cancer.

L73 ANSWER 83 OF 152 MEDLINE on STN DUPLICATE 39
AB **Sulfotransferase** 1A1 (**SULT1A1**) (thermostable **phenol sulfotransferase**, TS PST1, P-**PST**) is important in the metabolism of thyroid hormones. **SULT1A1** isolated from human platelets displays wide individual variations not only in the levels of activity, but also in thermal stability. The activity of the **allelic variant** or **allozyme** **SULT1A1***1, which possesses an arginine at amino acid position 213 (Arg213) has been shown to be more thermostable than the activity of the **SULT1A1***2 **allozyme** which possesses a histidine at this position (His213) when using p-nitrophenol as the substrate. We isolated a **SULT1A1***1 cDNA from a human liver cDNA library and expressed both **SULT1A1***1 and **SULT1A1***2 in eukaryotic cells. The **allozymes** were assayed using iodothyronines as the substrates and their biochemical properties were compared. **SULT1A1***1 activity was more thermostable and more sensitive to NaCl than was **SULT1A1** *2 activity when assayed with 3,5,3'-triiodothyronine (T(3)). Sensitivities to 2,6-dichloro-4-nitrophenol (DCNP) and apparent K(m) values for **SULT1A1***1 and for **SULT1A1***2 with iodothyronines were similar. Based on K(m) values, the preferences of these **SULT1A1** **allozymes** for iodothyronine substrates were the same (3,3'-diiodothyronine (3,3'-T(2))>3', 5',3-triiodothyronine (rT(3))>T(3)>thyroxine (T(4))>>3,5-diiodothyronine (3,5-T(2))). **SULT1A1***1 activity was significantly higher than the **SULT1A1***2 activity with T(3) as the substrate. Potential differences in thyroid hormone sulfation between individuals with predominant **SULT1A1***1 versus **SULT1A1***2 **allozymes** are most likely due to differences in catalytic activity rather than substrate specificity.

L73 ANSWER 86 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB A review on the interindividual variability of several forms of human **sulfotransferases** (STs). Five forms have been characterized for their substrate specificities, thermal stability, inhibitor sensitivity and regulation. Three forms are grouped under the expression **phenol**-STs. Two of these forms are thermostable and their diagnostic substrate is 4-nitrophenol, while the third form is thermolabile and its diagnostic substrate is dopamine. There is a form of hydroxysteroid ST, designated dehydroepiandrosterone ST, and its diagnostic substrate is dehydroepiandrosterone. There is also a form of estrogen ST whose diagnostic substrate is estrone or estradiol.

L73 ANSWER 87 OF 152 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
AB N-acetyltransferases (NAT), sulfotransferases (SULT) and heterocyclic amine activation in the mamma were investigated. Human mammary tissues were obtained from Caucasian women undergoing reduction mammoplasty. Mammary cytosols were prepared from human mammary tissue. mRNA was isolated from both human mammary tissue and human mammary epithelial cells. Due to the complexity of the NAT1 gene **polymorphism**, several polymerase chain reaction-based methods (using specific DNA primers) were used to determine NAT1 and NAT2 genotypes. Experimental observations gained from use of enzyme cofactors and NAT- and/or SULT-inhibitors on cytosolic enzyme activity, recombinant NAT1 activity and heterocyclic amine-DNA adduct formation suggest that both NAT1 and **SULT1A** enzymes contribute significantly to the activation of N-hydroxylated heterocyclic amines in mammary tissue. NAT1 mRNA levels were found to be 2- to 3-fold higher than mRNA transcripts of the NAT2 gene in reduction mammaoplasty tissue and mammary epithelial cells. NAT1-specific p-aminobenzoic acid acetylation activity, but not NAT2-specific sulfamethazine acetylation activity, was detectable in mammary cytosols. (92 ref)

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L73 ANSWER 89 OF 152 MEDLINE on STN DUPLICATE 43
AB Single nucleotide **polymorphisms** (SNPs) in genes encoding drug-metabolizing enzymes, transporters, receptors, and other drug targets have been widely implicated as contributors to differences among individuals as regards the efficacy and toxicity of many medications, as well as the susceptibility to complex diseases. By combining the polymerase chain reaction (PCR) technique with direct sequencing, we screened genomic DNAs from 48 Japanese volunteers for SNPs in genes encoding three quinone oxidoreductases (NQO1, NQO2, and PIG3) and 17 sulfotransferases (**SULT1A1**, **SULT1A2**, **SULT1A3**, SULT1C1, SULT1C2, SULT2A1, SULT2B1, ST1B2, TPST1, TPST2, SULTX3, STE, CST, HNK-1 ST, CHST2, CHST4, and CHST5). In all, we identified 320 SNPs from these 20 loci: 22 within coding elements, 21 in 5' flanking regions, 10 in 5' untranslated regions, 223 in introns, 19 in 3' untranslated regions, and 25 in 3' flanking regions. The ratio of transitions to transversions was approximately 2.3 to 1. Of the 22 coding SNPs, 6 were nonsynonymous substitutions that resulted in amino-acid substitutions. The high-density SNP maps we constructed from this data for each of the quinone oxidoreductases and sulfotransferases examined here should provide useful information for investigations designed to detect association(s) between genetic variations and common diseases or responsiveness to drug therapy.

L73 ANSWER 90 OF 152 MEDLINE on STN DUPLICATE 44
AB This commentary was written to stimulate thoughts on, and consideration of, enzyme expression data in target organs when investigating possible associations between **polymorphisms** in carcinogen activation enzymes, lifestyle/dietary factors and cancer risk. The lung and breast are taken as examples. There is overwhelming evidence for a genotoxic mechanism in lung cancer development, and compelling evidence for the

contribution of genotoxins to breast cancer aetiology. A consistent association has been shown where lung cancer risk is decreased by a G-->A **polymorphism** in the myeloperoxidase (MPO) gene, which is expressed in neutrophils recruited to the lung after chemical or immunological insults. In the breast, a consistent lack of association has been observed for women who are fast N:-acetyltransferase type 2 (NAT2) acetylators consuming cooked meat. This could be explained by the lack of detectable NAT2-associated sulfamethazine acetylation activity in cytosols prepared from mammary tissue, suggesting a minor contribution to carcinogen activation. The recent identification in mammary cytosols of detectable sulfotransferase isoforms (**SULT1A1** and **SULT1A3**), which have high catalytic efficiency for activating N:-hydroxylated heterocyclic amines (HCAs, **mutagens** in cooked meat), offers a more important role for these enzymes in the metabolic activation of genotoxins in the breast. The possible contribution of MPO and lactoperoxidase enzymes to carcinogen activation in mammary tissue is also considered. Sulfotransferases and peroxidases have wide substrate specificity in terms of carcinogen activation (HCAs, aromatic amines and polycyclic aromatic hydrocarbons-all present in cooked meat and tobacco smoke) compared with NATs (HCAs and aromatic amines only). For gene-environment interactions, investigations into functional **polymorphisms** in SULT and peroxidase genes may, therefore, offer new evidence for the involvement of genotoxins in the initiation of carcinogenesis. Identification of the isoforms (if any) of carcinogen activation enzymes that are expressed in the organs of interest will help to determine which genes to investigate in these studies.

L73 ANSWER 92 OF 152 MEDLINE on STN DUPLICATE 45

AB Sulfotransferase (SULT) 1A1 is involved in the inactivation of estrogens and bioactivation of heterocyclic amines and polycyclic aromatic hydrocarbons. A G--->A transition at codon 213 (CGC/Arg to CAC/His) of the **SULT1A1** gene was reported recently, and individuals homozygous for the His **allele** have a substantially lower activity of this enzyme than those with other genotypes. We hypothesized that the His **allele** may be a risk factor for breast cancer, particularly among women who had risk factors related to higher endogenous estrogen exposure. This hypothesis was investigated in a case-control study conducted in a cohort of postmenopausal Iowa women who completed a mailed questionnaire in 1986 on lifestyle factors including information on major breast cancer risk factors. DNA samples and information related to well-done meat intake were obtained from breast cancer cases diagnosed between 1992 and 1994 and a random sample of cancer-free cohort members. Multivariate analysis was performed on data from 156 cases and 332 controls who donated a blood sample. The frequency of the His **allele** was 41.6% in cases and 34.1% in controls (P = 0.03), and the risk of breast cancer was increased with the number of His **alleles** (P for trend = 0.02). Compared with women with the Arg/Arg genotype, an 80% elevated risk was observed among women homozygous for the His **allele** (95% confidence interval, 1.0-3.2; P = 0.04). This positive association was more pronounced among women who drank alcohol and had a high body mass index, early age at menarche, and late age at menopause, factors related to high endogenous estrogen exposure, than among those who did not have these risk factors. The risk of breast cancer was elevated with increasing doneness level of red meat intake among women with the Arg/Arg genotype (P for trend, 0.01) or the Arg/His genotype (P for trend, 0.10), whereas this association was not evident for women with the His/His genotype. The results from this study suggest that homozygosity for the **SULT1A1** His213 **allele** may be a risk factor for breast cancer, and its effect may be modified by the exposure level of endogenous estrogens and heterocyclic amines.

L73 ANSWER 94 OF 152 MEDLINE on STN DUPLICATE 46

AB Sulfotransferase (SULT) enzymes catalyze the sulfate conjugation of drugs, other xenobiotics, neurotransmitters and hormones. The genes for **SULT1A1** and **SULT1A2** contain common genetic

polymorphisms that are associated with individual variations in levels of enzyme activity as well as variations in biochemical and physical properties. We set out to compare the frequencies of common **SULT1A1** and **SULT1A2 alleles** in Caucasian, Chinese and African-American subjects. **Allele** frequencies for **SULT1A1***1, *2 and *3 in 242 Caucasian subjects were 0.656, 0.332 and 0.012, respectively. Frequencies of those same **alleles** were significantly different in 290 Chinese subjects: 0.914, 0.080 and 0.006, respectively, as were frequencies in 70 African-American subjects: 0.477, 0.294 and 0.229, respectively. Ethnic variation in **allele** frequencies was also observed for **SULT1A2**, with frequencies in Caucasian subjects for **SULT1A2***1, *2 and *3 of 0.507, 0.389 and 0.104; frequencies in Chinese of 0.924 and 0.076 with no *3 **alleles** observed; and, finally, in African-Americans frequencies of 0.637, 0.249 and 0.114, respectively. We also found that **SULT1A1***2 and **SULT1A2***2, the most common **variant** **alleles** for these two genes, were in positive linkage disequilibrium in all three populations studied, with D' values of 0.776 in Caucasian ($P < 0.001$), 0.915 in Chinese ($P < 0.001$) and 0.864 in African-American subjects ($P < 0.001$). These observations represent a step towards determining the possible functional implications for individual variations in sulfate conjugation of common genetic **polymorphisms** for **SULT1A1** and **SULT1A2**.

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L73 ANSWER 96 OF 152 MEDLINE on STN DUPLICATE 47
AB Cytosolic sulphotransferases transfer the sulpho moiety from the cofactor 5'-phosphoadenosine-3'-phosphosulphate (PAPS) to nucleophilic groups of xenobiotics and small endogenous compounds (such as hormones and neurotransmitters). This reaction often leads to products that can be excreted readily. However, other sulpho conjugates are strong electrophiles and may covalently bind with DNA and proteins. All known cytosolic sulphotransferases are members of an enzyme/gene superfamily termed SULT. In humans, 10 SULT genes are known. One of these genes encodes two different enzyme forms due to the use of alternative first exons. Different SULT forms substantially differ in their substrate specificity and tissue distribution. Genetic **polymorphisms** have been described for three human SULTs. Several **allelic** **variants** differ in functional properties, including the activation of promutagens. Only initial results are available from the analysis of SULT **allele** frequencies in different population groups, e.g. subjects suffering from specific diseases and corresponding controls.

L73 ANSWER 99 OF 152 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
AB An isolated nucleic acid molecule consisting of a **SULT1A3** sequence which contains a DNA sequence **variant**, is new. Also claimed are: isolated nucleic acids containing **SULT1A1** and **SULT1A2** sequences, where the sequences contain a DNA sequence **variant**; a method for determining the predisposition to a hormone dependent disease in a patient which involves detecting the presence or absence of a sulfotransferase DNA sequence **variant**; a method for determining sulfonator status in a patient by detecting the presence or absence of a sulfotransferase **allozyme**; an antibody with specific binding affinity for a sulfotransferase; a nucleic acid construct consisting of a **SULT1A3** DNA sequence operably linked to regulatory sequences; and an article consisting of a substrate and an array of different sulfotransferase nucleic acid molecules immobilized on the substrate. The above DNA sequences may be useful for detecting the presence of hormone-dependent disease such as cancers, especially mamma, prostate and ovary cancer. . (38pp)

L73 ANSWER 102 OF 152 MEDLINE on STN DUPLICATE 51
AB In recent years, significant effort has been made to identify genes that

influence breast cancer risk. Because the high-penetrance breast cancer susceptibility genes BRCA1 and 2 play a role only in a small fraction of breast cancer cases, understanding the genetic risk of the majority of breast cancers will require the identification and analysis of several lower penetrance genes. The estrogen-signaling pathway plays a crucial role in the pathophysiology of breast cancer; therefore, **polymorphism** in genes involved in this pathway is likely to influence breast cancer risk. Our detailed analysis of gene expression profiles of estrogen- and 4-OH-tamoxifen-treated ZR75-1 breast cancer cells identified members of the **sulfotransferase** 1A (**SULT1A) phenol sulfotransferase** family as downstream targets of tamoxifen. On the basis of the induction of **SULT1A** by 4-OH-tamoxifen and the known inherited variability in **SULT1A** enzymatic activity, we hypothesized that **polymorphism** in **sulfotransferase** genes might influence the risk of breast cancer. Using an RFLP that distinguishes an arginine to histidine change in exon 7 of the **SULT1A1** gene, we characterized **SULT1A1** genotypes in relation to breast cancer risk. An analysis of 444 breast cancer patients and 227 controls revealed no effect of **SULT1A1** genotype on the risk of breast cancer (P = 0.69); however, it did appear to influence the age of onset among early-onset affected patients (P = 0.04). Moreover, individuals with the higher activity **SULT1A1***1 **allele** were more likely to have other tumors in addition to breast cancer (P = 0.004; odds ratio, 3.02; 95% confidence interval, 1.32, 8.09). The large number of environmental **mutagens** and carcinogens activated by **sulfotransferases** and the high frequency of the **SULT1A1** *1 **allele** in human populations warrants additional studies to address the role of SULT genes in human cancer.

L73 ANSWER 104 OF 152 MEDLINE on STN DUPLICATE 53

AB Sulfate conjugation by **sulfotransferase** enzymes is an important pathway for the detoxication of xenobiotics and endogenous compounds. The large surface area of the gastrointestinal tract exposes the body to a range of potential toxins, and hence local metabolism is likely to be important. The ability of different regions of the gut to sulfate micromolar concentrations of simple **phenols** and catecholamines has been determined throughout the gut using 4-nitrophenol and dopamine as standard substrates. The pattern of sulfation of both compounds was similar, with activity highest in the small bowel >right colon >left colon >rectum >stomach >esophagus. High concentrations of **sulfotransferases** in the reservoir areas of the right and left colon indicate possible importance in detoxication by sulfation and also perhaps in activating **mutagens** in the same areas. Nutritional factors, such as a high-fat diet may, however, alter **sulfotransferase** activity.

L73 ANSWER 105 OF 152 MEDLINE on STN DUPLICATE 54

AB In a previous study of nine human breast-derived cell lines, rates of metabolism of 17beta-estradiol (E(2)) were greatly enhanced when cultures were exposed to the aromatic hydrocarbon receptor agonist, 2,3,7,8-tetrachlorodibenzo-p-dioxin. Elevated rates of E(2) hydroxylation at the C-2, -4, -6alpha and -15alpha positions were observed concomitant with the induction of cytochromes P450 1A1 and 1B1. In each cell line, 2- and 4-hydroxyestradiol (2- and 4-OHE(2)) were converted to 2- and 4-methoxyestradiol (2- and 4-MeOE(2)) by the action of catechol O:-methyltransferase. In this study, conjugation of these estrogen metabolites was investigated. A comparison of the levels of metabolites determined with and without prior treatment of the media with a crude beta-glucuronidase/sulfatase preparation showed that most of the 2-MeOE(2) present was in conjugated form, whereas 4-MeOE(2), 6alpha-OHE(2) and 15alpha-OHE(2) were minimally conjugated. Inhibitor studies suggested that it was the sulfatase activity of the preparation that hydrolyzed the 2-MeOE(2) conjugates in MCF-7 cell media; the presence of 2-MeOE(2)-3-sulfate in MCF-7 culture media was confirmed by electrospray

ion-trap mass spectrometry. To identify the enzyme catalyzing this conjugation, the expression of mRNAs encoding five sulfotransferases (**SULT1A1**, **SULT1A2**, **SULT1A3**, SULT1E1 and SULT2A1) was evaluated in the nine cell lines by use of the reverse transcription-polymerase chain reaction. Only expression of **SULT1A1** mRNA correlated with the observed conjugation of nanomolar levels of 2-MeOE(2) in these cell lines. Cloning and sequencing of **SULT1A1** cDNA from MCF-7 cells revealed that mRNAs encoding two previously identified **allelic variants**, **SULT1A1***1 ((213)Arg) and **SULT1A1***2 ((213)His), were expressed in these cells. Heterologous cDNA-directed expression of either **variant** in MDA-MB-231 cells, which do not normally express **SULT1A1**, conferred 2-MeOE(2) sulfonation activity. The **SULT1A1 allelic variants** were also expressed in SF:9 insect cells, from which post-microsomal supernatants were used to determine K:(m) values of 0.90 +/- 0.12 and 0.81 +/- 0.06 microM for **SULT1A1***1 and **SULT1A1***2, respectively, with 2-MeOE(2) as substrate. These results show that **SULT1A1** is an efficient and selective catalyst of 2-MeOE(2) sulfonation and, as such, may be important in modulating the anticarcinogenic effects of 2-MeOE(2) that have been described recently.

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L73 ANSWER 111 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

AB A thermostable **phenol sulfotransferase**, **SULT1A1**, has been implicated in numerous detoxification and bioactivation pathways; however, little is known regarding its endogenous function or its putative role in mediating risk for human environmental disease. A simple endpoint colorimetric assay is described that can be used for rapid phenotyping of **SULT1A1** activity in human populations. The assay utilizes a microtiter-plate format and relatively small amounts of platelet cytosol-derived enzyme. The enzyme catalyzes the synthesis of 2-naphthylsulfate from 2-naphthol and 5'-phosphoadenosine 3'-phosphosulfate (PAPS), whereas addition of p-nitrophenyl sulfate to the assay contributes to an effective PAPS-regenerating system. In contrast to other **sulfotransferase** assay methods, 3'-phosphoadenosine 5'-phosphate (PAP) does not accumulate during the incubation to interfere with enzyme activity, but instead serves as a cofactor to cause the removal of sulfate from p-nitrophenyl sulfate to regenerate PAPS. This reaction concomitantly results in generation of p-nitrophenol that can be quantified colorimetrically at 405 nm (epsilon = 18,200 M-1) to give an indirect measure of **sulfotransferase** activity. Using platelet enzyme preparations from adult human subjects, sulfation rates of two prototypical thermostable **phenol sulfotransferase** substrates (2-naphthol and p-nitrophenol) and one thermolabile **phenol sulfotransferase** substrate (dopamine) were determined using standard radiochemical protocols. These data were then compared with results from the colorimetric assay using 2-naphthol as substrate. There was a good correlation between the phenotyping assay and radiochemical assays for both 2-naphthol **sulfotransferase** and p-nitrophenol **sulfotransferase** activity (r = 0.85 and 0.69, respectively). However, **SULT1A1** activity was approximately 10 to 20 times higher with the colorimetric determination. As anticipated, there was no correlation between **SULT1A1** activity and dopamine **sulfotransferase** activity (r = 0.07) in these human platelet preparations. This inexpensive and rapid method for phenotyping **SULT1A1** activity may help investigators assess a role for this enzyme in disease susceptibility.

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L73 ANSWER 114 OF 152 MEDLINE on STN DUPLICATE 56
AB Sulfation catalysed by human cytosolic **sulfotransferases** is generally considered to be a detoxification mechanism. Recently, it has been demonstrated that sulfation of heterocyclic aromatic amines by human **phenol sulfotransferase** (**SULT1A1**) can result in a DNA binding species. Therefore, sulfation capacity has the potential to influence chemical carcinogenesis in humans. To date, one genetic **polymorphism** (Arg213His) has been identified that is associated with reduced platelet **sulfotransferase** activity. In this study, data on age, race, gender, **SULT1A1** genotype and platelet **SULT1A1** activity were available for 279 individuals. A simple colorimetric phenotyping assay, in conjunction with genotyping, was employed to demonstrate a significant correlation ($r = 0.23$, $P < 0.01$) of **SULT1A1** genotype and platelet **sulfotransferase** activity towards 2-naphthol, a marker substrate for this enzyme. There was also a difference in mean **sulfotransferase** activity based on gender (1.28 nmol/min/mg, females; 0.94 nmol/min/mg, males, $P = 0.001$). DNA binding studies using recombinant **SULT1A1***1 and **SULT1A1***2 revealed that **SULT1A1***1 catalysed N-hydroxy-aminobiphenyl (N-OH-ABP) DNA adduct formation with substantially greater efficiency (5.4 versus 0.4 pmol bound/mg DNA/20 min) than the **SULT1A1***2 **variant.** A similar pattern was observed with 2-hydroxyamino-1-methyl-6-phenylimidazo[4,5b]pyridine (N-OH-PhIP) (4.6 versus 1.8 pmol bound/mg DNA/20 min).

L73 ANSWER 120 OF 152 MEDLINE on STN DUPLICATE 59
AB Preliminary evidence suggests that genetic **polymorphisms** in certain enzymes involved in xenobiotic metabolism and chemical defense could modify a susceptibility to prostate cancer. In the present study, two recently described **phenol** sulphotransferase **SULT1A1** **alleles** (**SULT1A1***1, **SULT1A1***2) were investigated using a polymerase chain reaction-restriction fragment length **polymorphism** (PCR-RFLP) approach. Genotyping was performed on DNA isolated from white blood cells from 134 patients with prostate cancer and 184 healthy control subjects. Both the prostate cancer patients and the controls demonstrated similar frequencies of the **variant** **allele** **SULT1A1***2 (35.1% vs 39.1%). Homozygosity for the **variant** **allele** was slightly less frequent in cancer patients than controls (12.7% vs 17.4%). Our study does not support the hypothesis that the **phenol** sulphotransferase **variant** **allele** **SULT1A1***2 with a G/A transition at nucleotide 638 is a risk modifier for prostate cancer in the Caucasian population.

L73 ANSWER 121 OF 152 HCAPLUS COPYRIGHT 2006 ACS on STN
AB A review with 27 refs. Three related forms of **phenol** **sulfotransferase**, thermostable ST1A2 and ST1A3 and a thermolabile ST1A5 are known to exist in human livers. Thermostable forms, whose activities are **polymorphically** distributed in human tissues, have been shown to mediate the bioactivation of carcinogenic N-hydroxy aromatic amines as well as **phenolic** substrates. **Variant** forms of ST1A3 mRNAs (i.e. Arg213His and Met223Val) have been found in human livers. In a Japanese population, **allele** frequencies of 213Arg and 213His were 0.83 and 0.17, resp. No remarkable difference in [35S]3'-phosphoadenosine 5'-phosphosulfate (PAPS)-dependent sulfation of p-nitrophenol was observed between recombinant 213Arg- and 213His-type ST1A3, although it was reported that 213His homozygosity was associated with both lower (less than one-sevenths) p-nitrophenol sulfation and thermolability in human platelets. The recombinant ST1A3 (213His) did exhibit unstability at 45° and 37° as compared with ST1A3 (213Arg). Liver cytosols from 213His homozygotes did not always show low p-nitrophenol-sulfating activities. Different mol. mechanisms in sulfation **polymorphism** are suggested in livers and platelets of humans.

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L73 ANSWER 123 OF 152 MEDLINE on STN DUPLICATE 60
AB Three human **phenol sulfotransferases**, provisionally named **SULT1A1**, 1A2 and 1A3, show 91-96% homology of their amino acid sequences and are encoded by neighbouring gene loci. Functional genetic **polymorphisms** are known for two of these **sulfotransferases**. In **SULT1A1**, a G to A transition leads to an Arg213 to His exchange and eliminates a Bsp143II restriction site. **SULT1A1***His shows lower enzyme activity and thermostability than **SULT1A1***Arg. In **SULT1A2**, an A to C transversion causes an Asn235 to Thr exchange and introduces a BpiI restriction site. Enzyme **SULT1A2***Thr is less active than **SULT1A2***Asn. These substitutions were detected by restriction fragment length **polymorphism** analyses of genomic sequences amplified by polymerase chain reaction. Despite the high similarity between the different human **SULT1A** genes, it was possible to amplify specifically the **polymorphic** parts of either **SULT1A1** or 1A2, but not the homologous sequences of the other SULT, by setting the forward primer into intron 6. DNA from 300 adult male Caucasian subjects was analysed. **Allele** frequencies were 0.63 and 0.37 for **SULT1A1***Arg and *His, and 0.62 and 0.38 for **SULT1A2***Asn and *Thr, respectively. The frequency of the haplotype **SULT1A1***Arg/**SULT1A2***Asn (0.61) was nearly as high as the **allele** frequencies of its components. The same was observed for the haplotype **SULT1A1***His/**SULT1A2***Thr, whose frequency was 0.35. In contrast, haplotypes 1A1*Arg/1A2*Thr and 1A1*His/1A2*Asn were very rare. Their frequencies (0.02 each) were less than 10% of the figures expected in an independent distribution. The results demonstrate a strong association of the **alleles** producing the more active enzyme **variants** (**SULT1A1***Arg and **SULT1A2***Asn) and of those encoding the less active **variants** (**SULT1A1***His and **SULT1A2***Thr).

L73 ANSWER 124 OF 152 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 61
AB Cytosolic **sulfotransferase** catalyzes sulfoconjugation of relatively small lipophilic endobiotics and xenobiotics. At least 44 cytosolic **sulfotransferases** have been identified from mammals, and based on their amino acid sequences, these forms are shown to constitute five different families. In humans, 10 **sulfotransferase** genes have been identified and shown to localize on at least five different chromosomes. The enzymatic properties characterized in the recombinant forms indicate the association of their substrate specificity with metabolisms of such nonpeptide hormones as estrogen, corticoid, and thyroxine, although most forms are also active on the sulfation of various xenobiotics. Genetic **polymorphisms** are observed on such human **sulfotransferases** as ST1A2, ST1A3, and ST2A3.

L73 ANSWER 125 OF 152 MEDLINE on STN DUPLICATE 62
AB Conjugation of xenobiotics is often associated with detoxification. However, this traditional view is one-sided. In particular, numerous compounds are known that are metabolized to chemically reactive metabolites via sulfation (O-sulfonation). This can be rationalized by the fact that the sulfate group is electron-withdrawing and may be cleaved off heterolytically in appropriate molecules, thus leading to the formation of a strongly electrophilic cation. The heterologous expression of sulfotransferases in indicator cells of standard **mutagenicity** tests has substantially improved the accessibility of this activation pathway. The use of this technology is important, since many reactive sulfate conjugates only show strong toxicological effects if they are generated directly within the indicator cell, due to their insufficient penetration of cell membranes. Xenobiotic-metabolizing sulfotransferases are cytosolic enzymes, which form a superfamily (SULT). Eleven distinct

human SULT forms are known, which strongly differ in their tissue distribution and their substrate specificity. Common functionally relevant genetic **polymorphisms** of the transcribed region are known for two of the forms, **SULT1A1** and 1A2. Studies using recombinant test systems demonstrate that many promutagens are activated with high selectivity by an individual SULT form. Pronounced differences in promutagen activation were detected between the different human forms, including their **allelic variants**, and also between orthologous SULTs from different species. Therefore, SULTs may be involved in the individual genetic disposition, species differences, and organotropisms for toxicological effects of chemicals. Activation by SULTs differs from other activation pathway in its cyclic nature: reaction of a sulfuric acid ester with water usually regenerates the hydroxylated compound, which becomes available for a new cycle of activation. SULT-mediated reactivation may even occur if another initial reactive species, e.g. an epoxide, has reacted with water.

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AB An isolated nucleic acid molecule (I) which contains an STP2 sequence **polymorphism**, not as part of a natural chromosome, is new. Also claimed are: DNA probes (II) for detecting STP2 locus **polymorphisms** which contain any 48 **polymorphic** sequences; an array of 2 or more (II); and a method for detecting a **polymorphism** in STP2 metabolism of a substrate by analyzing a genome for the 48 specified sequences, the presence of which indicates alterations in STP2 expression or activity. STP2 encodes a **phenol-sulfotransferase** which is involves in the metabolism of drugs such as minoxidil, acetaminophen, p-nitrophenol, etc. The DNA probes may be useful for screening and genotyping, i.e. for predicting the rate of metabolism of STP2 substrates, potential drug-drug interactions and adverse side-effects. They may also be useful for detecting disease which result from accidental exposure to toxins and for establishing animal models for drug metabolism. Also disclosed are proteins encoded by **polymorphisms** from STP2 and their recombinant production, transgenic animals containing the **polymorphisms** and assays for determining the effect of STP2 **polymorphism.** (45pp)

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AB **Phenol sulfotransferases** (PSTs or **phenol** SULTs) catalyze the sulfate conjugation of **phenolic** drugs, xenobiotics, and monoamines. Two human **PST** isoforms have been defined biochemically, a thermostable (TS), or **phenol** -preferring, and a thermolabile (TL), or monoamine-preferring form. Pharmacogenetic studies showed that levels of both TS **PST** activity and TS **PST** thermal stability (an indirect measure of variation in amino acid sequence) in the platelet were regulated by genetic **polymorphisms.** Subsequent molecular genetic experiments revealed the existence of three human **PST** genes, two of which, **SULT1A1** and **SULT1A2**, encode proteins with "TS **PST**-like" activity. We recently reported common nucleotide **polymorphisms** for **SULT1A1** that are associated with variations in platelet TS **PST** activity and thermal stability. In the present experiments, we set out to determine whether functionally significant DNA **polymorphisms** also might exist for **SULT1A2**, to compare the biochemical properties of all common

allozymes encoded by **SULT1A2** and **SULT1A1**, and to study **phenol** SULT genotype-phenotype correlations in the human liver. We phenotyped 61 human liver biopsy samples for TS **PST** thermal stability and activity. The open reading frames of **SULT1A2** and **SULT1A1** then were amplified with the polymerase chain reaction and sequenced for each of these hepatic tissue samples. We observed 13 **SULT1A2 alleles** that encoded 6 **allozymes.** These **alleles** were in linkage disequilibrium with **alleles** for **SULT1A1.** Biochemical characterization of common **allozymes** encoded by both genes suggested that **SULT1A1** was primarily responsible for "TS **PST** phenotype" in the human liver. In summary, both **SULT1A2** and **SULT1A1** have a series of common **alleles** encoding enzymes that differ functionally and are associated with individual differences in **phenol** SULT properties in the liver.

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AB Cytosolic **sulfotransferases** (ST) catalyze the sulfation of various **phenolic** agents, catecholamines, thyroid hormones, steroids, drugs, and procarcinogens, usually resulting in the inactivation and subsequent excretion of the compound. My laboratory's efforts have focused on the cloning of the human **phenol**-sulfating (**PST**) members of this gene superfamily, implicated in the bioactivation of the hair growth stimulant, minoxidil. At least two major forms of human **PST** enzymes have been characterized biochemically, the **phenol**-preferring **PST** (P-**PST**), and the catecholamine-preferring **PST** (M-**PST**). Various cDNAs have been cloned representing **alleles** of 3 gene loci termed as STP1, STP2, and STM, which were all mapped precisely to a small region on human chromosome 16p and to the homologous region of mouse chromosome 7. Human cosmid genomic clones have been sequenced to determine the genomic organization for each of the 3 highly-related genes. All contain 7 coding exons, with conserved intron-exon boundaries, and presumptive alternative tissue-specific promoters. At least one of the 3 **PST**-encoding genes is responsible for forming minoxidil sulfate in the lower outer root sheath of anagen hair follicles. The steroid sulfating genes, STD and STE, have been cloned by other laboratories. The isozyme products of these genes sulfate DHEA and estrogens, respectively. I hypothesize that either STE or STD is involved in the formation of cholesterol sulfate (CS) in epidermal keratinocytes. CS has been demonstrated by other groups to be an activator of keratinocyte Protein Kinase Ceta, which subsequently results in the activation of epidermal transglutaminase and formation of the cornified envelop. STE or STD might also be involved in bioinactivation of estrogens and androgens within skin. Our recent unpublished results have focused on elucidating the patterns of ST gene expression in cultured keratinocytes and fibroblasts derived from human skin using RT-PCR, to understand which of the 5 different ST genes in involved in the regulation of keratinocyte differentiation and minoxidil-induced hair growth.

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AB Sulphation, catalysed by members of the sulphotransferase (SULT) enzyme family, is an important component of the body's chemical defence mechanism, but also acts to bioactivate **mutagens** such as hydroxylated aryl and heterocyclic amines. A major human sulphotransferase, **SULT1A1** (P-**PST**), metabolizes and/or bioactivates many drugs, iodothyronines and hydroxylated aromatic amines. The enzyme activity varies widely within the population and is under genetic control. We have developed an assay detecting a G-->A transition in **SULT1A1** that causes an Arg213-->His substitution associated

with low SULT activity and altered enzyme properties, and have used it to assess the **SULT1A1** genotype in Caucasian (n=293) and African (Nigerian, n=52) populations. We show that the **mutant SULT1A1*2 allele** is present at frequencies of 0.321 and 0.269 in the Caucasian and African populations respectively. We also demonstrate a significant age-related difference in **SULT1A1** genotype within our Caucasian population, with increasing incidence of **SULT1A1***1 homozygosity and decreasing incidence of **SULT1A1** *2 homozygosity with increasing age, indicating a potential association of **SULT1A1***1 **allozyme**(s) with protection against cell and/or tissue damage during aging.

=> log y

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